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A BACTERIOLOGICAL AND CLINICAL STUDY OF THE PROFESSIONAL PERSONNEL OF MATERNITY HOSPITALS, WITH SPECIAL REFERENCE TO CARRIERS OF HÆMOLYTIC STREPTOCOCCI.

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AT the end of December, 1935, the authors were asked to investigate an outbreak of puerperal sepsis and to discuss with the medical officers of the hospital concerned the application of available bacteriological knowledge to the prevention of further spread or future occurrence of puerperal infection.

The salient facts revealed by a review of the current literature were as follows:

1. Of 4,655 deaths directly attributable to childbirth investigated in Great Britain, 38% were due to puerperal sepsis. Almost half (46%) of these deaths from sepsis followed normal deliveries; in other words, infection following normal labour caused 18% of all deaths in this series.[1]

2. It was estimated that not less than 85% of these fatal infections following normal labour were due to hæmolytic streptococci. These organisms were also responsible for a somewhat less but variable percentage of infections following instrumental or difficult labour.

3. A table given by Dora Colebrook[2] showed that the mortality of hæmolytic streptococcal infection, even under apparently optimal conditions of treatment at that time, was not less than 21% of

its incidence; the responsibility of this infection for many cases of prolonged illness or possibly permanent invalidism must also be considered.

4. Lancefield[3] divided hæmolytic streptococci into several groups (designated A, B, C, D and E, further groups being added later) by means of a precipitin test. Strains from various human infections almost invariably belonged to Group A; this test therefore afforded a method of distinguishing pathogenic human strains from those derived from other sources, including animal infections. Lancefield and Hare[4] later showed that almost all the infecting strains in a series of cases of severe puerperal sepsis fell into Group A. They also examined 855 women in the ante-natal period, from only 13 of whom they isolated hæmolytic streptococci. Among these 13 women there was one case of very mild puerperal pyrexia. Twelve of the 13 strains were studied, and none were found to belong to Group A. These authors also showed that Group A streptococci were extremely rare in the genital tract of women with afebrile puerperia or with minor puerperal infections. Earlier work reviewed by one of us,[5] which also indicated the rarity of hæmolytic streptococci in the human vagina under normal conditions, is thus confirmed.

5. On the other hand, hæmolytic streptococci have been found in the apparently normal human upper respiratory tract in 5% to 40% of various groups investigated. In one series of 100 strains from nurses, schoolboys and a few other adults, Hare[6] found that about one-third of these could be classified as Group A. If, as Hare suggests, the average incidence of hæmolytic streptococci in the throats of the normal population is assumed from the figures quoted above to be about 20%, it is probable therefore that some 7% will harbour pathogenic Group A strains. This work of Hare and that of Griffith,[7] who showed that the hæmolytic streptococci associated with various human diseases could be divided into at least twenty-seven serologically different types, has been amplified in connexion with puerperal sepsis in careful studies by J. Smith[8] and Dora Colebrook.[2] Both these workers used agglutination tests for the study of the relationship of the hæmolytic streptococci found in the genital tract, peritoneal cavity or blood stream in puerperal infections to those in the respiratory tract of the patients themselves and those of their contacts, familial and professional. Smith in 49 cases found streptococci identical with the infecting strain in the patient's own respiratory tract in 16·3% and in contacts' respiratory tracts in 63·3%, in a few cases in both, and in two other septic foci (one in a patient, one in a contact). His figures therefore established a possible source of infection in 83·7% of his cases. Dora Colebrook investigated 63 cases; she found streptococci identical with the infecting strain in the patient's respiratory tract in 38·1% and in the respiratory tract of contacts in 57·1%, and twice in other septic foci (one in a patient, one in a contact). These figures indicated a possible source of infection in 76·1% of her cases; but she pointed out that among the earlier cases in this series the examination (particularly of nasal swabs from contacts) was incomplete, and that if consideration was given only to those (31 in number) in which a full complement of swabs was obtained from both the patient and her contacts, the percentage in which a possible source of infection was found became 87.

Two outstanding facts therefore emerge in respect of the possibility of preventing the occurrence of spread of puerperal infection:

1. That, as nearly half the cases follow normal labour, "it is clear that neither improvement in ante-natal work nor increased dexterity or judgement in obstetric operations" (whatever may be their value in lowering the maternal death or morbidity rate from other causes) "will have any appreciable effect on this moiety of the fatalities, but that we must look for improvement in other directions".[1]

2. That as evidence is accumulating that the upper respiratory tract of the patient herself, or more frequently that of her contacts, is the most probable source of puerperal infection with hæmolytic streptococci (after either normal or abnormal delivery), attention must be directed to possible methods of preventing transfer of organisms from these sites to the genital tract immediately prior to, during and after childbirth.

With these facts in mind, the investigation of the outbreak of sepsis referred to above was commenced. This outbreak occurred in the intermediate wards of a community hospital. The nursing staff consisted of permanent day and night sisters and staff nurses, and a changing personnel of trainee nurses. The sequence of events was briefly as follows:

Mrs. A. was delivered on December 6, 1935. She developed pyrexia on December 14, 1935. Blood was taken on December 30, 1935, but no growth was reported on attempted culture. The course of the illness continued to be clinically septicæmic until the patient's death on January 31, 1936.

Mrs. B. was delivered on December 11, 1935. Pyrexia occurred on December 18, 1935. No growth was reported from an attempted blood culture on January 19, 1936. The patient recovered after five weeks' illness.

Mrs. C. was delivered on December 27, 1935. A blood culture made by one of us (P.T.) on December 30, 1935, yielded a growth of hæmolytic streptococci. The patient died on January 8, 1936.

Each of these patients was under the care of a different private medical attendant, and therefore, although in each case the necessary precautions had been taken and the regulations complied with individually, the possible connexion between the three cases was not emphasized until their occurrence became known to the medical committee in control of the hospital, who immediately recognized the gravity of the situation.

Inquiry into possible sources of infection revealed that Mrs. A. had been attended from December 6

to 12, 1935, by a nurse (number 9) who became ill on the latter date with acute tonsillitis. We isolated a scanty growth of Group A streptococci from this nurse's throat on January 2, 1936. Mrs. B. was in the same room as Mrs. A. until the latter developed pyrexia. Mrs. C. was said to have had "quinsy" about fourteen days before delivery. In her case this illness may have been the source of her septicæmia, but it is also possible that it was due to transference of organisms from contacts of the earlier cases who were possibly harbouring hæmolytic streptococci.

We were not able, owing to lack of opportunity to make bacteriological investigations, to prove that Mrs. A. or Mrs. B. was infected with hæmolytic streptococci. Nevertheless the sequence of events, the clinical histories and the isolation of hæmolytic streptococci from a nurse recently recovered from tonsillitis, who had nursed Mrs. A., and from the third patient, Mrs. C., made it probable that this organism was responsible for the illness in each case. If this was so, there had been abundant opportunity for dissemination of hæmolytic streptococci among the nurses who had had contact with one or more of the patients and with each other.

It was therefore decided to take swabs from the nose and throat of all the professional contacts of the three patients. These contacts numbered 32 in all.

The technique of the bacteriological procedures is fully described elsewhere, but it may be stated here that swabs which yielded a growth of hæmolytic streptococci in the primary culture on blood agar plates are referred to as positive, and those from which they were absent as negative. An approximate estimate of the number of colonies of hæmolytic streptococci on plates from positive swabs was attempted, the results of which have been recorded in this paper as follows:

Less than 12 colonies, scanty (+) growth.
13 to 50 colonies, fairly profuse (++) growth.
Numerous colonies (often predominating), profuse (+++) growth.
An apparently pure culture, pure (++++) growth.

Hæmolytic streptococci were found in nasal swabs (taken from the anterior nares) in four (2·1% of the 149 individuals swabbed, and in each case the throat swabs were also positive to the same or a greater degree. Subsequent descriptions in this paper refer, therefore, to throat swabs only.

At this time we could not identify Group A strains, since sera of the type described by Lancefield were not available; we considered that the biochemical methods used by Hare and Colebrook[9] to distinguish pathogenic strains would take too long to be helpful in our immediate practical problem. It was therefore apparent that the safest course in the meantime would be to act on the results of the direct cultures on blood agar plates, on which provisional reports could be made on the day after the swabs were taken. These cultures showed that 15 (47%) of the 32 contacts had positive swabs; in eight of these the growth was profuse or fairly profuse, in spite of the absence at this time of obvious upper respiratory tract infection in any of the individuals concerned.

It was evident, therefore, that the carrier rate among the nursing staff was abnormally high, even assuming, on the basis of Hare's work,[6] that only one-third of the strains isolated would be shown by fuller investigation to be of pathogenic type. Withdrawal from duty of those with positive swabs, or alternatively only those with profuse or fairly profuse growths, would have seriously depleted the staff. It was decided, therefore, that in spite of the unwelcome notoriety and financial loss involved, the most certain way to prevent immediate spread and to safeguard future patients would be to refuse further admissions, and as soon as practicable to close the hospital pending further bacteriological and clinical investigations of the carriers among the nursing staff, and thorough disinfection of the building.

The hospital was reopened in February, 1936. At this time there were no carriers of hæmolytic streptococci among the senior permanent staff, with the exception of one sister (number 18). Her swabs frequently showed hæmolytic streptococci, which, however, were not classified as Group A. It was realized, however, that it would not be practical, even if possible, to ensure that no carriers existed among the staff, especially among the temporary trainees. It appeared that the best means of minimizing the risk of infection of patients from possible nose and throat "reservoirs" in attendants would be to systematize a scheme for the conduct of the midwifery wards, which stressed this danger and provided for methods of preventing it as far as lay within the power of the hospital staff.

The following is a brief outline of the scheme which was devised.

A. Procedures concerning the patient herself:
  (1) During the ante-natal period:
    (a) Inquiry into history of upper respiratory tract infections with bacteriological investigations and treatment if necessary.
    (b) Instruction of patient regarding risk involved by contact with persons suffering from coughs, "colds", sore throats, septic sores *et cetera*, and emphasis of the necessity to report the existence of such conditions in herself.
  (2) After admission:
    (a) Inquiry into history again if necessary, and treatment of any existing respiratory tract or septic infections.
    (b) Instructions similar to those given in the ante-natal period, but with special reference to visitors.
    (c) Detailed teaching regarding personal hygiene, including the use of handkerchiefs (paper ones recommended), talking, laughing, sneezing *et cetera*.
    (d) Disinfection of hands (as suggested by Dora Colebrook[10]), especially before the use of towels, utensils *et cetera*.

B. Procedures concerning contacts after patient's admission to hospital:

(1) Visitors:

(a) Limitation of numbers, and instructions regarding loud talking, laughing, coughing, kissing *et cetera*.

(b) Absolute prohibition of visitors suffering from "colds" *et cetera*.

(2) Professional attendants:

(a) Lectures and demonstrations to nurses, explaining and amplifying the instructions given to patients and visitors.

(b) Directions to nurses to report immediately the existence of sore throats, "colds", septic fingers *et cetera* in patients or themselves.

(c) Adoption of measures, such as masks, antiseptic ritual *et cetera*, especially designed to prevent transference of organisms from nose and throat reservoirs by droplets expelled during expiration, or in dried secretion on fingers, handkerchiefs, towels *et cetera*.

(d) Maintenance of surgical asepsis in regard to utensils, instruments, dressings, gowns, gloves *et cetera*.

(e) Preparation of the patient. It is recommended that the perineal toilet be carried out by a method such as that described by Colebrook and Maxted.[10]

(f) Restriction of vaginal examinations.

(g) Periodic swabbing of the throats of the staff.

The value and practicability of routine periodic swabbing (which is not to be confused with swabbing undertaken in connexion with outbreaks of streptococcal infection) was considered doubtful. It certainly serves to impress on nurses the paramount importance of the nose and throat as sources of puerperal infection, even if only occasionally carried out, and it may reveal the presence of an unsuspected carrier of pathogenic streptococci. On the other hand, unless it can be done very frequently, almost daily, a procedure which would require enormous laboratory facilities, it gives no guarantee of freedom from streptococci in the intervening periods; lack of appreciation of this latter point may even lead to a false sense of security when "negative" reports are received, and perhaps a tendency to rely on the results of swabs as safeguards rather than on unremitting careful attention to the details of the various other measures designed to prevent transference of hæmolytic streptococci from the nose and throat to the genital tract of the patient.

Notwithstanding the limitations of periodic swabbing as a preventive measure, we considered, in view of the extremely high incidence of hæmolytic streptococci among the staff revealed by the investigations described above, that it would be both advisable and of interest to carry out further swabbing at times when there was no associated sepsis among the patients, and to examine the relationship, if any, between the bacteriological and clinical findings. Notes were made of the history as regards upper respiratory infection, and of the clinical condition of those from whom swabs were taken. Persons with doubtful infections and those in whom treatment appeared to be indicated as a result of the bacteriological and preliminary clinical examinations were referred to Dr. Jean Littlejohn, honorary oto-rhino-laryngologist to the Queen Victoria Hospital, to whom we are much indebted for her help and advice.

The material used in this study is therefore derived from:

1. The patient Mrs. C. and the 32 contacts involved in the outbreak of puerperal sepsis described above.

2. A second group of 67 members of the resident medical and nursing staff connected with the midwifery department of the same community hospital and the associated public hospital whose swabs were taken during May and June, 1936.

3. A third group of 74 similar individuals whose swabs were taken during September and October, 1936.

(Some of the permanent members of the staff were included in more than one of these groups, the actual number of individuals swabbed being 131.)

4. Eighteen medical practitioners and nurses engaged in midwifery in other hospitals. This group includes contacts of the patients referred to in this paper as Mrs. D. (suffering from fulminating puerperal sepsis, probably but not proven streptococcal), Mrs. E. and Mrs. F. (both with proven streptococcal infections).

The members of these four groups were at the time of swabbing in normal health or in health regarded by themselves as sufficiently good to enable them to work. Swabs taken in connexion with illnesses such as tonsillitis sufficiently severe to cause incapacitation have not been included, as such conditions constitute a different problem from that of the apparently healthy midwifery worker, who nevertheless harbours potentially pathogenic streptococci in the respiratory tract.

The results may be considered under two headings: the bacteriological investigations and the correlation of bacteriological and clinical findings.

Bacteriological Investigations.

Technical Methods.

Swabs were taken from the throat and anterior nares. It has been shown[11] that if separate swabs are taken from the tonsillar fossæ and posterior naso-pharynx (the latter with a guarded swab to avoid mouth contamination), the organisms may differ either in kind or in predominance. The duplication of swabs is, however, a serious consideration when dealing with large numbers, and a single swab may be used, provided it is brought into firm contact successively with the tonsils or tonsillar fossæ and the walls of the posterior naso-pharynx. Swabs from the anterior nares were taken with a swab, moistened with sterile broth, which was passed up to the mucous membrane between the blades of a sterilized nasal speculum.

All swabs were plated within six hours on 10% horse blood agar plates, which were incubated aerobically. Fry[12] has demonstrated more marked

hæmolysis by means of anaerobic cultures with a few streptococci isolated from puerperal infections. Hare[6] therefore used anaerobic culture for the primary plates in his series of throat swabs. It is not known, however, as far as we are aware, what percentage of hæmolytic streptococci from throat cultures would show this phenomenon if comparisons were made between aerobic and anaerobic cultures. In view, therefore, of the small number of such strains encountered by Fry or by one of us (L.M.B.) in a previous investigation,[13] it was not considered necessary to employ the relatively more time-consuming anaerobic method in this investigation. A few true hæmolytic strains may thus have been missed, but, on the other hand, some strains which were very doubtfully hæmolytic on the blood agar plates were regarded provisionally as such and were isolated for further examination.

The approximate number of hæmolytic streptococcal colonies on individual plates is shown in Tables I and III.

Biochemical tests were carried out by the methods described by Hare and Colebrook.[9]

It was suggested by Dr. F. M. Burnet that sensitivity to bacteriophage might prove useful in the comparison of the various strains isolated. Four serological types of streptococcal bacteriophage described,[14] and designated as types A, B, C and D by Miss Alice Evans, of the United States Public Health Service, to whom we are greatly indebted, were obtained from her and used in this investigation. Two methods of testing sensitivity were used: (i) Demonstration of lysis in broth cultures; a few drops of an overnight culture in Wright's pneumococcus broth of the strain to be tested was added to each of two tubes of the same broth. After incubation for 30 to 40 minutes at 37° C. a drop of lytic filtrate was added to one tube, the other remaining as a control. The tubes were incubated overnight, and complete or partial clearing (that is, a turbidity definitely less than that in the control tube) was regarded as evidence of lysis. (ii) Demonstration of plaques on agar plates: a few drops of an overnight broth culture were spread evenly on a suitably dry 1% agar plate. After incubation for one hour at 37° C. a drop of lytic filtrate was spread over the culture, which was then incubated overnight. The next day the plate was examined for the presence of an area of complete lysis or, in the case of lesser sensitivity, for confluent or individual plaques. The results of the two methods were in the main parallel. Alice Evans considers the test-tube method the more sensitive, but we found it easier to demonstrate the presence of a few plaques than to assess the value of small differences in turbidity. Lytic filtrate was used in preference to phage in the nascent state (that is, in the presence of a known sensitive strain in addition to the one being tested), as the more widespread action of the latter, described by Evans, would appear to militate against its usefulness in the differentiation of strains.

Precipitin Tests.—Lancefield's type strains were not available when this work was begun. In view of her findings and their specific confirmation in respect of the streptococci present in most cases of severe puerperal sepsis, it was considered justifiable to proceed with the immunization of rabbits with the strain isolated from the blood stream of Mrs. C. For purposes of comparison, sera were also prepared from strain 35 (Evans's strain 563, sensitive to phage B) and from strain 24. This strain grew on blood agar plates in a pure culture of colonies with the matt surface suggestive of virulence; it was isolated from a case of acute tonsillitis. The method of preparation of immunizing emulsions described by Lancefield was followed in detail. Three series of injections were given, the interval between each series being three days. Each injection in the first series consisted of 1·0 cubic centimetre of a 1 in 20 dilution of the final emulsion; in the second, of 2·0 cubic centimetres of the same dilution; in the third series an injection of 0·5 cubic centimetre of a 1 in 5 dilution was followed by two injections of 1·0 cubic centimetre of this dilution. All injections were given intravenously.

The rabbits were bled for testing five days after the last injection, and for supplies of serum five days later. Extracts of the organisms to be tested were prepared and the precipitin tests were performed by Lancefield's method with the homologous strains and subsequently with others from various human sources.[1] The results are shown in Tables I and II. A reaction regarded as conforming to the written description of that due to the presence in the extract of the group-specific "C" substance consisted in the development of a thick ring of precipitate at the junction of the layers of serum and extract. This was usually obvious within ten minutes at room temperature, but was occasionally intensified during the earlier part of the subsequent two hours' immersion in a water bath at 37° C. It then gradually dispersed, and after overnight refrigeration was visible as a thick disk-like deposit on the bottom of the tube. Occasionally thin disk-like deposits were present after overnight refrigeration in tubes in which there had been no previous "ring" precipitates. These are probably similar to the non-specific deposits described by Lancefield.[3] Traces of fine granular deposit were occasionally observed after overnight refrigeration, especially in tests performed by the micro-method described by Lancefield and Hare.[4]

[1] The sera prepared by us have been tested by Mr. D. Murnane, B.V.Sc., of the Council of Science and Industry at the Veterinary Research Institute, Melbourne, with extracts of strains received from Dr. Lancefield. We are greatly indebted to Mr. Murnane for the performance of these tests and for permission to report them. His results, using the ring test as the criterion, show clear-cut group specificity, which placed our strains 1 and 24 in Group A, and strain 35 in Group C. Organisms giving only these two latter types of reaction were not placed in Group A. The precipitin tests were performed at first by Lancefield's method, by the micro-method just mentioned, and finally in the majority of cases by a macroscopic method, proportionately similar but smaller volumes (0·1 and 0·2 cubic centimetre of serum and extract respectively) than those used by Lancefield being used.

TABLE I.

Biochemical, Precipitin and Bacteriophage Reactions of Streptococci from Case of Puerperal Sepsis (Mrs. C.) and her Contacts.

Strain Number.	Source.	Soluble Hæmolysin.	Final pH.	Growth on Bile Agar.		Hydrolysis of Sodium Hippurate.	Fermentation Tests.					Precipitin Tests.			Sensitivity to Bacteriophage.			
				10%.	40%.		Lactose.	Mannite.	Salicin.	Sorbite.	Trehalose.	Serum. 1.	Serum. 24.	Serum. 35.	Phage. A.	Phage. B.	Phage. C.	Phage. D.
1	Blood (Mrs. C.)	+++	5·4	0	0	0	+++	0	+++	0	+	+++	+++	0	br. +++ ag. +++	0	0	0
2	Throat (+++) Nurse 25.	+++	5·5	0		0	+++	0	+++	0	0	0			br. ++ ag. +	0	0	0
3	Throat (+) Nurse 61.	+++	4·9	0		0	+++	0	+++	0	+++	++			br. + ag. ++	0	0	0
4 (i)	Throat (+) Nurse 9.	+++	5·4	0		0	+++	0	++	0	+	+			br. +++ ag. ++	0	0	0
(ii)		+++	4·9	0		+++	+++	+++	+++	+	0	0			0	0	0	0
5	Throat (++) Nurse 54.	+++	5·6	0		+	+++	0	+++	0	0	0			br. +++ ag. +	0	0	0
6	Throat (+) Nurse 16.	+++	5·4	0		0	+++	0	+++	0	++	++			br. ++ ag. +	0	0	0
7	Throat (++) Nurse 13.	+++	5·5	0		0	+++	0	+++	0	+	++			br. ++ ag. ++	0	0	0
14	Throat (+++) Nurse 13.	+++								0	+	+			ag. +++	0	0	0
8	Throat (+) Nurse 40.	+++	5·0	+	±	++	+++	0	+++	+	+	0			0	br. + ag. ++	0	0
9	Throat (++) Nurse 5.	0	4·9	0		+++	+++	0	+++	0	0	0			0	0	0	0
10	Throat (+) Sister 18.	+++	5·0	0		+++	+++	0	+++	0	+++	Trace.	0	0	0	0	0	0
13	Throat (+++) Sister 18.	+++								0	+++	Trace.	0	0	0	0	0	0
32	Throat (++) Sister 18.									0	+++	Trace.	0	0	0	0	0	0
11	Throat (++) Staff Nurse 150.	+	4·8	0		0	+++	0	+++	0	+++	0			0	0	0	0
18	Throat (+) Staff Nurse 150.	+++					+++	0	+++	0	0	+++			br. +++ ag. +++	0	0	0
12	Throat (+) Sister 7.	0	4·7	0		+++	+++	0	+++	0	+++	0			0	0	0	0
17	Throat (++) Sister 7.	+++					++	0	++	0	+	+			ag. +	0	0	0

(+) = scanty growth (1 to 12 colonies) of streptococci regarded as hæmolytic on primary blood agar plate cultures.
(++) = fairly profuse growth (13 to 50 colonies) of streptococci regarded as hæmolytic on primary blood agar plate cultures.
(+++) = predominating growth (more than 50 colonies) of streptococci regarded as hæmolytic on primary blood agar plate cultures.
+++ = strongly positive biochemical or precipitin reactions or complete lysis by bacteriophage.
++ or + = less strongly positive biochemical or precipitin reactions or incomplete lysis by bacteriophage.
0 = negative biochemical or precipitin reactions or absence of lysis by bacteriophage.
br. = broth.
ag. = agar

TABLE I.—*Continued.*

Biochemical, Precipitin and Bacteriophage Reactions of Streptococci from Case of Puerperal Sepsis (Mrs. C.) and her Contacts.—Continued.

Strain Number.	Source.	Soluble Hæmolysin.	Final pH.	Growth on Bile Agar.		Hydrolysis of Sodium Hippurate.	Fermentation Tests.					Precipitin Tests.			Sensitivity to Bacteriophage.			
				10%.	40%.		Lactose.	Mannite.	Salicin.	Sorbite.	Trehalose.	Serum. 1.	Serum. 24.	Serum. 35.	Phage. A.	Phage. B.	Phage. C.	Phage. D.
15	Throat (+) Nurse 109.	+								0	+	0			0	ag. +	0	0
16	Throat (+) Nurse 108.	0													br. ++ ag. ++	0	0	0
19	Throat (+) Nurse 130.									0	+++	0			0	0	0	0
20	Throat (+) Nurse 123									0	+	+++			0	br. ++ ag. +	0	0

(±) = scanty growth (1 to 12 colonies) of streptococci regarded as hæmolytic on primary blood agar plate cultures.
(+) = fairly profuse growth (13 to 50 colonies) of streptococci regarded as hæmolytic on primary blood agar plate cultures.
(++) = predominating growth (more than 50 colonies) of streptococci regarded as hæmolytic on primary blood agar plate cultures.
(+++) = strongly positive biochemical or precipitin reactions or complete lysis by bacteriophage.
++ = less strongly positive biochemical or precipitin reactions or incomplete lysis by bacteriophage.
+ or 0 = negative biochemical or precipitin reactions or absence of lysis by bacteriophage.
br. = broth.
ag. = agar.

Analysis and Discussion of Bacteriological Findings.

The strain "Mrs. C." and those isolated from 15 of the first group of individuals were subjected to the biochemical, bacteriophage and precipitin tests described above, with the results shown in Table I. The biochemical tests placed 10 of the 15 throat strains in the human pathogenic group. Later, 7 of these 10 strains gave a positive result to the precipitin test. We therefore considered that 7 (22%) of the 32 contacts harboured Group A streptococci.

The bacteriophage and precipitin tests were also carried out with 16 other strains from various human streptococcal diseases, the results of which are shown in Table II. From these results and those shown in Table I it will be seen that while the majority of Group A strains were sensitive to phage A, the agreement was not absolute; two strains not placed in Group A were lysed by phage A, while a few Group A strains were insensitive to any of the four races of phage or were lysed by races other than A. It was therefore obvious that sensitivity to the four available types of phage could not be used as a quick method of identifying the Group A strains. On the other hand, the variation of susceptibility to phage among the strains falling into Lancefield's Group A suggests that bacteriophage differentiation might be of value in more detailed epidemiological studies.

These findings indicate, therefore, that unless a quicker satisfactory method supplants it, the precipitin test remains the best means of distinguishing potentially pathogenic hæmolytic streptococci from less harmful types. It was therefore the method of choice for the differentiation of the hæmolytic streptococci isolated from the primary plate cultures of swabs from our second, third and fourth groups of persons.

In the second group, 12 individuals were found to harbour hæmolytic streptococci, of which two (3%) were classified as Group A.

In the third group, hæmolytic streptococci were isolated from 18; five of these fell into Group A, making its incidence in this group 6·7%.

The fourth group is not considered as a whole, as the swabs were not taken under comparable conditions.

Correlation of Bacteriological and Clinical Findings.

It is obvious that, apart from the general significance (which is discussed below) of the varying percentages of positive swabs obtained at these different times, each individual must be considered separately if the results of swabbings are to play any part in determining his or her infectivity to midwifery patients. In this connexion we soon realized, as Dora Colebrook has also pointed out, that more reliance must be placed on the results of multiple swabbings (especially if repeated at short intervals) than on those of a single swab, particularly when the culture from it is "negative". But it was not always possible to obtain more than one swab, especially from the trainees; for purposes

TABLE II.

Results of Precipitin and Bacteriophage Tests with Hæmolytic Streptococci from various Human Sources.

Strain Number.	Source.	Precipitin Test.			Sensitivity to Bacteriophage.			
		Serum.			Phage.			
		1.	24.	35.	A.	B.	C.	D.
1	Puerperal septicæmia	+++	+++	0	br. +++ ag. +++	0	0	0
21	Acute tonsillitis	+++			br. + ag. +++	0	0	0
22	Cellulitis	+++			0	0	0	0
23	Abscess	0			0	br. +++ ag. +++	0	0
24	Acute tonsillitis	+++	+++	0	0	0	0	0
25	Pleural fluid	+++			0	0	0	0
26	Acute tonsillitis	+++			0	0	0	0
27	Subcutaneous abscess	++			0	0	0	0
28	Fæces (in case of acute rheumatic fever)	+++			0	0	0	0
29	Urine (pyelitis)	+			br. 0 ag. +	0	0	0
30	Urine (pyelitis)	0			0	0	0	0
33	*Otitis media*	+++			br. +++ ag. +++	0	0	0
34 Evans 751/A	Sore throat	+++			br. +++ ag. +++	0	0	0
35 Evans 563/B	? Erysipelas ("Birkhaug E.1" strain) ? Empyema	0	0	+++	0	br. +++ ag. +++	0	0
36 Evans 594/C	Scarlet fever	0			0	0	br. +++ ag. +++	0
37 Evans 646/C	Scarlet fever	+++			0	0	br. +++ ag. +++	0
38 Evans 693/D	Fæces (case of intestinal hæmorrhage)	0			0	0	0	br. ++ ag. +++

+++ = strongly positive precipitin test, or complete lysis by bacteriophage.
++ or + = less strongly positive precipitin test, or incomplete lysis by bacteriophage.
0 = negative precipitin test, or absence of lysis by bacteriophage.
br. = broth.
ag. = agar.

TABLE III.

Individual Relationship of Results of Swabbing to Clinical Findings and Known Contact with Streptococcal Infections.[1]

Case Number.	Date and Result of Swabs.	Day (D.) or Night (N.) Duty.	Known Recent Contact with Streptococcal Infection.	Condition of Upper Respiratory Tract.	Comments.
9. Nurse.	2/1/36. A.+	D.	Mrs. A. (? streptococcal).	Infected tonsils.	Nursed Mrs. A. till she went off duty with tonsillitis on 8/12/35.
16. Nurse.	31/12/35. A.+	D.	Mrs. C.	Infected tonsils.	
24. R.M.O.	25/5/36. +	D. and N.	Nil.	Normal.	
25. Nurse.	2/1/36. +++	D.	Mrs. C.	Infected tonsils.	"Influenza" and tonsillitis 8–27/12/35.
40. Nurse.	31/12/35. +	D.	Mrs. C.	Normal.	
48. Sister.	3/2/37. A.++	N.	Mrs. D. (? streptococcal).	Infected tonsils.	
61. Nurse.	2/1/36. A.+	D.	Mrs. C.	Normal.	
100. Nurse.	3/6/36. +	D.	Nil.	Normal.	
131. Sister.	3/2/37. A.+	D.	Mrs. D.	Normal.	
144. R.M.O.	25/5/36. +	D. and N.	Surgical septic cases.	Normal.	
5. Nurse.	31/12/35. ++ 9/2/36. 0	D. D.	Mrs. C. Nil since Mrs. C.	Normal.	
47. Sister.	5/8/36. + 10/8/36. 0	N. N.	Nil. Nil.	Normal.	
91. Nurse.	3/6/36. + 5/8/36. 0	D. D.	Nil. Nil.	Normal.	
134. Nurse.	13/8/36. 0 24/8/36. ++	D. N.	Nil. Nil.	Normal.	
21. R.M.O.	26/5/36. A.+ 5/8/36. 0	D. and N. D. and N.	Surgical septic cases. Surgical septic cases.	Infected tonsils. Very frequent "colds".	
63. Nurse.	28/5/36. 0 17/8/36. U.+++	D. N.	Cases of septic abortion. Cases of septic abortion.	Infected tonsils. Frequent "colds".	The culture from this nurse's swab was accidentally destroyed before the precipitin test was performed.
130. Nurse.	5/2/36. + 5/8/36. 0	D. D.	Nil. Nil.	Normal.	
13. Staff nurse.	31/12/35. A.++ 4/2/36. A.++	D. D.	Mrs. C. None since Mrs. C.	Normal.	
75. Sister.	3/2/37. A.++ 15/2/37. A.++	N. Off duty.	Mrs. D.	Normal.	This sister went off duty following the death of Mrs. D. on 4/2/37.
95. Nurse.	3/8/36. A.++ 10/8/36. A.++	N. N.	Cases of septic abortion. Cases of septic abortion.	Normal.	
123. Nurse.	5/2/36. A.+ 24/8/36. +	D. D.	Mrs. C. Nil.	Normal.	

A indicates that the streptococcus concerned gave a positive Group A precipitin test.
U indicates that the streptococcus concerned was not classified by means of the precipitin test
All other streptococci were tested, but did not belong to Group A.

[1] Cases in which all swabs were negative (unless four or more) are not included in this table, but are tabulated collectively in Table IV.

TABLE III.—*Continued.*

Individual Relationship of Results of Swabbing to Clinical Findings and Known Contact with Streptococcal Infections.[1]—Continued.

Case Number.	Date and Result of Swabs.	Day (D.) or Night (N.) Duty.	Known Recent Contact with Streptococcal Infection.	Condition of Upper Respiratory Tract.	Comments.
7. Sister.	31/12/35. 0 2/1/36. 0 4/2/36. A.++	D. D. D.	Mrs. C. Mrs. C. None since Mrs. C.	Normal.	
54. Staff nurse.	31/12/35. A.++ 1/6/36. 0 19/8/36. 0	D. D. D.	Mrs. C. Nil. Nil.	Normal.	
90. Nurse.	28/5/36. + 5/8/36. 0 31/8/36. 0	D. D. D.	Nil. Nil. Nil.	Normal.	
108. Nurse.	4/2/36. 0 3/6/36. +++ 8/9/36. 0	D. D. and N. D.	Nil. Nil.	Normal.	The positive swab was taken on the day after return from district nursing.
82. District sister.	2/3/36. A.+++ 3/8/36. 0 12/10/36. 0	D. and N. D. and N. D. and N.	Nil. Nil. Nil.	Infected tonsils.	Tonsillectomy performed late in March, 1936.
133. Nurse.	29/8/36. +++ 31/8/36. 0 3/9/36. 0	D. D. D.	Nil. Nil. Nil.	Infected tonsils.	
92. Sister.	3/6/36. 0 10/8/36. A.+++ 13/8/36. A.+	D. D. Off duty.	Nil. Nil. Nil.	Normal.	
46. District sister.	15/6/36. A.++ 3/8/36. A.++ 12/10/36. 0	D. and N. D. and N. D. and N.	Nil. Nil. Nil.	Infected tonsils.	Tonsillectomy performed in September, 1936 (after swabbing).
60. Nurse.	2/6/36. ++ 3/6/36. ++ 15/6/36. +++	D. D. D.	Nil. Nil. Nil.	Infected tonsils.	
15. Nurse.	1/6/36. + 3/6/36. ++ 30/6/36. 0 12/8/36. + 19/8/36. 0 8/9/36. + 15/9/36. ++	N. N. D. N. D. N. N.	Nil. Nil. Nil. Nil. Nil. Contact Mrs. G. 3–5/9/36. None since 5/9/36.	Normal.	
18. Sister.	2/1/36. + 31/1/36. + 4/2/36. +++ 24/3/36. ++	D. D. D. D.	Mrs. C. None since 8/1/36. None since 8/1/36. None since 8/1/36.	Frequent "colds". Antral infection suspected.	
66. Nurse.	12/8/36. ++ 19/8/36. + 24/8/36. ++ 3/9/36. 0	N. Off duty. Off duty. D.	Nil. Nil. Nil. Nil.	Frequent "colds".	
68. Nurse.	10/8/36. ++++ 12/8/36. + 24/8/36. + 3/9/36. 0	N. Off duty. N. D.	Nil. Nil. Nil. Nil.	Recent acute tonsillitis.	
70. Sister.	2/1/36. 0 4/2/36. 0 29/4/36. 0 5/8/36. 0 8/9/36. 0	N. N. N. N. N.	Mrs. C. None since 8/1/36. None since 8/1/36. None since 8/1/36. None since 8/1/36.	Normal.	
71. Nurse.	26/5/36. A.+ 13/8/36. + 17/8/36. + 24/8/36. ++ 31/8/36. + 3/9/36. ++ 7/9/36. +	D. N. N. N. N. D. D.	Nil. Nil. Nil. Nil. Nil. Nil. Nil.	Large infected tonsils.	Tonsillectomy undertaken at end of September, 1936. Nurse did not subsequently return to hospital.

A indicates that the streptococcus concerned gave a positive Group A precipitin test.
U indicates that the streptococcus concerned was not classified by means of the precipitin test.
All other streptococci were tested, but did not belong to Group A.

[1] Cases in which all swabs were negative (unless four or more) are not included in this table, but are tabulated collectively in Table IV.

TABLE III.—*Continued*

Individual Relationship of Results of Swabbing to Clinical Findings and Known Contact with Streptococcal Infections.[1]—Continued.

Case Number.	Date and Result of Swabs.	Day (D.) or Night (N.) Duty.	Known Recent Contact with Streptococcal Infection.	Condition of Upper Respiratory Tract.	Comments.
81. Sister.	5/8/36. A.+ 10/8/36. A.+ 17/8/36. 0 7/9/36. A.+ 10/9/36. A.+	D. D. D. D. D.	Nil. Nil. Nil. Mrs. G. 3–5/9/36. None since 5/9/36.	Recent severe coryza. ? Sinusitis.	
84. Nurse.	1/6/36. 0 5/8/36. 0 8/9/36. +++ 28/9/36. +++ 30/9/36. +++	D. N. Off duty. D. Off duty.	Nil. Nil. Mrs. G. 3–5/9/36. None since 5/9/36. None since 5/9/36.	Severe coryza a week before swab taken on 8/9/36.	Streptococci not culturally similar to Mrs. G.'s strain.
88. Nurse.	27/5/36. 0 3/8/36. 0 31/8/36. 0 3/9/36. +++ 8/9/36. ++ 15/9/36. ++ 21/9/36. ++	D. D. D. D. Off duty. Off duty. Off duty.	Nil. Contact with scarlet fever and septic abortion till 25/8/36. Mrs. G. 3–5/9/36. None since 5/9/36. None since 5/9/36. None since 5/9/36.	Chronic nasopharyngitis. Tonsils enlarged.	Streptococci present 3–21/9/36 culturally similar to Mrs. G.'s strain.
96. Nurse.	2/6/36. 0 19/8/36. + 7/9/36. + 10/9/36. +	D. D. D. D.	Nil. Nil. Mrs. G. 3–5/9/36. None since 5/9/36.	Normal.	Streptococci present, not similar culturally to Mrs. G.'s strain.
109. Nurse.	4/2/36. + 3/6/36. 0 6/8/36. + 8/9/36. +	D. D. D. D.	Mrs. C. Nil. Nil. Mrs. G.	Normal.	Streptococci not culturally similar to Mrs. G.'s strain.
117. Nurse.	3/6/36. + 13/8/36. 0 31/8/36. 0 6/9/36. 0	D. D. D. D.	Nil. Nil. Nil. Mrs. G. till 5/9/36.	Normal.	
118. Nurse.	26/5/36. 0 10/8/36. ++++ 17/8/36. + 19/8/36. +++	D. D. Off duty. Off duty.	Nil. Nil. Nil. Nil.	Normal. Recent "influenza".	Returned for duty on 10/8/36 after "influenza".
120. Staff nurse.	29/4/36. 0 2/6/36. 0 12/8/36. 0 8/9/36. 0 15/9/36. 0	D. D. D. D. D.	Nil. Nil. Nil. Mrs. G. till 5/9/36. None since 5/9/36.	Normal.	
135. Sister.	10/5/37. A.+++ 25/5/37. A.+++ 2/6/37. A.+ 29/6/37. 0 2/7/37. 0 5/7/37. 0	D. and N. Off duty. Off duty. Off duty.	Contact Mrs. E. and Mrs. F. Mrs. F.	Acute coryza. Chronic nasopharyngitis. Enlarged infected tonsils. Slight nasopharyngitis.	Tonsillectomy 9/6/37.
136. Nurse.	26/5/36. 0 6/8/36. + 13/8/36. + 19/8/36. 0	D. D. D. D.	Nil. Contact with Nurse 118. Nil. Nil.	Frequent "colds". Slight tonsillitis (1–6/9/1936.)	
140. Sister.	26/5/36. ++ 12/8/36. + 19/8/36. ++ 31/8/36. 0	D. D. D. D.	Nil. Nil. Nil. Nil.	Frequent "colds". Infected tonsils and sinuses.	Has been a diphtheria carrier several times after contact.
143. Nurse.	5/2/36. 0 26/5/36. 0 12/8/36. + 19/8/36. ++ 24/8/36. ++	D D. D. Off duty. Off duty.	Mrs. C. Nil. Nil. Nil. Nil.	Quiescent antral infection. Quiescent antral infection. Active antral infection. Active antral infection. Active antral infection.	

A indicates that the streptococcus concerned gave a positive Group A precipitin test.
U indicates that the streptococcus concerned was not classified by means of the precipitin test.
All other streptococci were tested, but did not belong to Group A.

[1] Cases in which all swabs were negative (unless four or more) are not included in this table, but are tabulated collectively in Table IV.

TABLE III.—*Continued.*

Individual Relationship of Results of Swabbing to Clinical Findings and Known Contact with Streptococcal Infections.[1]—Continued.

Case Number.	Date and Result of Swabs.	Day (D.) or Night (N.) Duty.	Known Recent Contact with Streptococcal Infection.	Condition of Upper Respiratory Tract.	Comments.
150. Staff nurse.	31/12/35. ++ 4/2/36. A + 29/4/36. 0 6/8/36. ++ 17/8/36. + 3/9/36. 0	D. D. D. D. Off duty. D.	Mrs. C. None since 8/1/36. None since 8/1/36. None since 8/1/36. None since 8/1/36. None since 8/1/36.	Normal.	
139. Nurse.	3/8/36. A.++ 10/8/36. A.++ 17/8/36. A.+++ 24/8/36. A.+ 26/8/36. A.+ 31/8/36. A.+	D. Off duty. Off duty. Off duty. Off duty. Off duty.	Recent contact with scarlet fever.	Tonsils very large and infected.	Tonsillectomy 1/10/36. Nurse did not subsequently return to hospital.

A indicates that the streptococcus concerned gave a positive Group A precipitin test.
U indicates that the streptococcus concerned was not classified by means of the precipitin test.
All other streptococci were tested, but did not belong to Group A.
[1] Cases in which all swabs were negative (unless four or more) are not included in this table, but are tabulated collectively in Table IV.

of comparison, therefore, the individuals have been grouped according to the number of swabs taken:

1. Seventy-five individuals were swabbed once; from these, 10 positive cultures were obtained. One of the latter is of interest.

It was taken from a nurse (number 25) on December 31, 1935, following contact with Mrs. C. It showed a profuse growth of hæmolytic streptococci, which, however, when subsequently tested, did not give a Group A precipitin reaction. This nurse gave a history of having been warded for "influenza" and tonsillitis from December 8 to 27, 1935. After recovery she was examined by a rhinologist, who did not think that she required tonsillectomy.

This opinion, together with the diagnosis of "influenza", suggests that she did not suffer from typical acute follicular tonsillitis, after which one would expect to find Group A streptococci in a positive swab. We cannot exclude the possibility that a mixture of strains was present and that the one selected for the precipitin test happened not to belong to Group A. This possibility is inherent in bacteriological procedures in which one or more colonies are taken as representative of a greater number. In practice it is largely offset by the discernment, acquired by experience, of suggestive

TABLE IV.

Relationship of Results of Swabbing with Clinical Condition and Known Recent Contact with Hæmolytic Streptococcal Infections.

Number of Swabs Taken and Results of Culture.	Total Number of Persons Swabbed.	No Signs or Symptoms of Infection of Upper Respiratory Tract.						Signs and/or Symptoms of Chronic or Recent Acute Infection of Upper Respiratory Tract Present.					
		Swabs Negative.		Swabs Positive.				Swabs Negative.		Swabs Positive.			
				Group A Streptococci.		Not Group A.				Group A Streptococci.		Not Group A.	
		Total.	C.[1]	Total.	C.	Total.	C.	Total.	C.	Total.	C.	Total.	C.
1	75	58	24	2	2	4	2	7	2	3	3	1	1
2—both negative ..	23	20	5	—	—	—	—	3	0	—	—	—	—
2—1 positive	5	—	—	—	—	5	1	—	—	—	—	—	—
2—both positive.. ..	4	—	—	4	4	—	—	—	—	—	—	—	—
3—all negative	13	10	1	—	—	—	—	3	0	—	—	—	—
3—1 positive	6	—	—	2	2	2	1	—	—	1	0	1	0
3—2 positive	2	—	—	1	0	—	—	—	—	1	0	—	—
3—all positive	1	—	—	—	—	—	—	—	—	—	—	1	0
4 to 7—all negative ..	2	2	1	—	—	—	—	—	—	—	—	—	—
4 to 7—1 positive ..	0	—	—	—	—	—	—	—	—	—	—	—	—
4 to 7—2 positive ..	3	—	—	—	—	2	2	—	—	—	—	1	0
4 to 7—3 positive ..	10	—	—	—	—	1	1	—	—	1	1	8	3
4 to 7—all positive ..	5	—	—	1	1	1	1	—	—	2	1	1	0
Total	149	90	31	10	9	15	8	13	2	8	5	13	4

[1] Known recent contact with streptococcal infection.

cultural differences between colonies isolated from a primary plate. Nevertheless it would certainly be advisable for further swabs to be taken before a nurse with this history and with such a profuse growth of hæmolytic streptococci in her throat swab be allowed to resume midwifery duty.

2. Individuals swabbed twice numbered 34. Both swabs were "negative" in 25. Of interest among the latter are:

Number 8.—This sister was in charge of a ward containing surgical septic cases.

Number 93.—This nurse suffered from frequent "colds", on account of which she had had her tonsils removed and was under treatment for chronic antral infection. Swabs taken at the end of three months on night duty and after contact with Mrs. G. (see below) both showed no hæmolytic streptococci. Although two swabbings do not provide conclusive evidence, the negative results suggest that the antral infection in this nurse was not streptococcal and that she did not easily become a carrier of these organisms. It is of interest in her case that masking caused severe wheezing, possibly by accentuating an allergic sensitivity to her own naso-pharyngeal flora. Incidentally it would seem that an individual on whom the prolonged masking demanded in midwifery imposed such undue hardship would be better advised to concentrate on other branches.

Number 62.—This nurse, whose tonsils appeared healthy on examination, gave a history of "tonsillitis" during the week before her first swab was taken; it was noted during her illness, however, that there was no exudate of the type characteristic of acute follicular tonsillitis, and a swab taken at this time (not included in our series) was stated to have grown staphylococci only. It may be noted that this case and that of Nurse 25, described above, provide two instances of faucial infection which, after being recognized clinically as differing from typical follicular tonsillitis, were found bacteriologically not to be associated with Group A streptococci. In the absence of bacteriological control, however, the only safe course would be to assume that such tonsillitis, even if atypical, might be associated with the presence of streptococci infective to others.

Number 106.—This nurse stated that she suffered from frequent "colds" while on night duty. Two swabs taken during one of these "colds" were both "negative".

3. Twenty-two individuals were swabbed three times, in thirteen of whom all swabs were "negative". Three of those with "negative" swabs had definite clinical evidence of chronic infection of the upper respiratory tract, which was apparently not associated with streptococcal invasion.

4. Twenty were swabbed four or more times. Those calling for special comment are the following:

Number 18.—This sister was in the midwifery department during the outbreak of sepsis. Two scanty and two fairly profuse growths of hæmolytic streptococci were present in four swabs taken during January, February and March, 1936. The streptococci from each of these swabs gave similar traces of precipitate overnight with Group A serum, which were not regarded as group specific; these strains also did not show the sensitivity to phage A which characterized the patient's strain. This sister's streptococci could not therefore be considered to be associated with the outbreak of sepsis; their persistence, however, may have been related to her history of frequent "colds" and the clinical finding of large infected tonsils; an antral infection was also suspected, but she left hospital before further investigations could be undertaken.

Number 88.—This nurse had three "negative" swabs, the last of which was taken the day after cessation of contact with a patient with septic abortion (from whom cultures were not available). It was desired to exclude the possibility of infection from this source, as this nurse was due for a period in the midwifery ward. She was allowed to go to the midwifery ward a week later, and came into contact with a patient (Mrs. G.) who on the third day of her puerperium developed a mild pyrexia which lasted three days. Cultures from her *cervix uteri* on September 4, 1936, the second day of pyrexia, showed a profuse growth of large moist hæmolytic streptococcal colonies, associated in anaerobic culture with *Clostridium welchii*. The streptococci gave the fermentation reactions of *Streptococcus fæcalis* (Holman) and did not react with Group A serum, resembling in these respects the "pseudo-hæmolytic" type described by Hare and Maxted.[135] Strains isolated from the throat of Nurse 88 from swabs taken on October 3, 8, 15 and 21 were all similar culturally to those isolated from Mrs. G. and did not belong to Group A. It seems most likely that this nurse became a carrier through contact with Mrs. G., whose infection was probably a mild endogenous one following a difficult instrumental delivery. It is possible, however, that this nurse's streptococci were derived from the case of septic abortion with which she had previously been in contact, and if so, subsequent events showed that we were unwise in allowing her to proceed to midwifery work so soon after cessation of contact with the septic case on the unreliable evidence of one "negative" swab.

Number 135.—This sister was a contact of two cases (Mrs. E. and Mrs. F.) which occurred in a small private hospital. Mrs. E. developed pyrexia on April 24, 1937 (the third day of her puerperium); she was transferred to the Women's Hospital for isolation on May 1, 1937, and was still there, slowly recovering, three months later. Mrs. F. became pyrexial on May 5, 1937, the second day of the puerperium, and was sent to the Women's Hospital on May 7, 1937. Her infection proved fatal. Hæmolytic streptococci were isolated from both cases at the Women's Hospital, and were classified as Group A by Miss H. Butler, of the Baker Research Institute (personal communication). One of us (L.M.B.) was asked to take swabs from Sister 135 and the three other professional attendants of these two patients (two private medical practitioners and another sister) on May 10, 1937. The three last mentioned had "negative" swabs, but Sister 135 showed a profuse growth of Group A streptococci. She had a fairly severe "cold" at this time, but could not remember the exact date of its onset. She stated that she suffered from frequent "colds". Another profuse growth of Group A streptococci was present on May 25, 1937, and a scanty one on June 2, 1937. Her tonsils were enlarged and unhealthy. In the hope that it would reduce the likelihood of positive swabs interfering with her work in the future, she asked for tonsillectomy, which was performed on June 9, 1937. Swabs were taken on June 29, 1937, July 2, 1937, and July 5, 1937, all of which were "negative".

Number 139.—This nurse was a prospective midwifery trainee when her first swab was taken on August 3, 1936. She had recently been in contact with a case of scarlet fever. Her swabs repeatedly showed a fairly profuse growth of Group A streptococci. She stated that she rarely suffered from "colds" or "sore throats", but her tonsils were very large and appeared heavily infected. Tonsillectomy was performed on October 1, 1936. There was no opportunity to take swabs subsequently.

Number 140.—This sister stated that she had several times been found to be a temporary carrier of *Corynebacterium diphtheriæ* during outbreaks of diphtheria necessitating swabbing of contacts. She also gave a history of frequent "colds" and "sore throats" and of antral infection. An antral wash-out on October 5, 1937, was stated to have grown staphylococci only. Hæmolytic streptococci, not belonging to Group A, were found in her throat swabs on three occasions. There was no known recent contact with septic cases at the times when these swabs were taken, but the clinical history suggests that in the event of such contact this individual would be very likely to become a carrier of pathogenic streptococci.

Number 150.—This nurse's swabs gave anomalous results in that streptococci not belonging to Group A were found during contact with Mrs. C., while a Group A strain was isolated at another time when there was no known contact with sepsis.

The impression was prevalent among the nurses swabbed that positive findings were most frequent in those on night duty. A few individuals did show fairly profuse growths of hæmolytic streptococci while on night duty and "negative" swabs during alternating periods of day duty, without any correlated contact with sepsis. It is possible that night work in itself may favour the growth of hæmolytic streptococci, but this series is not large enough to provide evidence on this point.

The relevant details concerning every case in which one or more positive swabs were obtained are given in Table III; but the above discussion of certain of them, and of some in which all swabs were "negative", illustrates the variation in the findings, bacteriological and clinical, which may occur from person to person, and thus serves to emphasize the need for full consideration of each individual from all aspects before action is based on the results of swabbings.

On the other hand, some facts of general value emerge from an analysis of the figures as a whole. It will be seen from Table IV (which is based on the results of all the swabs taken, both "negative" and positive) that in 115 individuals with clinically healthy respiratory tracts, Group A streptococci were present in ten (8·7), in nine of whom (that is, in 90% of their incidence) there was known contact with hæmolytic streptococcal disease. In 34 persons with clinical evidence of chronic or recent acute infection of the upper respiratory tract, Group A strains were found in eight (23%) and were associated with contact in two of these (25%) of their incidence). Hæmolytic streptococci other than Group A strains were present in 15 (13%) of the healthy group and in 13 (38·3%) of those with clinical abnormality. Correlation with contact cannot be made in the cases of the heterogenous streptococci not belonging to Group A.

These figures indicate that when the naso-pharynx is normal contact may play an important part in determining both the presence and the duration of the carrier condition. Abnormality of the tissues in this area, as Shibley, Hanger and Dochez have also suggested,[16] presumably provides an environment more favourable than that of the healthy naso-pharynx for both the invasion by and persistence of hæmolytic streptococci, either as primary or secondary factors in a disease process. When their growth is thus favoured, hæmolytic streptococci acquired by contact do not disappear so readily on its cessation, and it may be found that "negative" swabs can be obtained only after treatment of the underlying abnormality.

It appears, therefore, that in general a considerable proportion of persons with a history or clinical evidence of abnormality of the naso-pharynx will be likely to harbour hæmolytic streptococci, perhaps in large numbers, at any time, whereas those with healthy respiratory tracts are most likely to have positive swabs during or for a short time after acute streptococcal illness or contact with such illness in others. But acute infections, even when trivial, are usually easily recognized, and, owing to the persistent teaching of Colebrook and others, their potential danger to puerperal patients is becoming more widely appreciated, with the result that provision is being more generally made for the carrying out of appropriate preventive measures, including temporary withdrawal of infected persons from midwifery work.

The chronic carrier, however, presents a lesser but more insidious danger. In this connexion we have not been unmindful of the difficulties created by recommendations, as a result of swabbing, that various members of the staff be withdrawn: administration and training are disorganized; the knowledge of positive swabs may cause mental distress in conscientious and imaginative individuals; occasionally passive resistance or resentment is shown, and a few individuals leave a hospital where strict precautions are taken, to become perhaps more dangerous in less controlled institutions; serious economic hardship may be imposed on a nurse who is forced to relinquish her work while undergoing treatment for a period which may run into several months.

These difficulties merit sympathy and practical help, but they cannot be allowed to outweigh consideration for the safety of the patient. There is no doubt that persons with acute infections or with swabs showing large numbers of hæmolytic streptococci should refrain from midwifery; but in the case of the chronic carrier of a few hæmolytic streptococci one is tempted to think (particularly if the organisms do not belong to Group A) that she is unlikely to transfer an infecting dose to the patient, and that, provided proper masking and other precautions are taken, the all-round efficiency of the midwifery unit may be best maintained by retaining her, especially if she is an experienced and reliable worker. On the other hand, such a senior member of the staff probably has the most prolonged and intimate contact with the patients and the most widespread opportunity to disseminate organisms among her colleagues.

The results of our investigation suggest that the risk of infection from the chronic carrier could be greatly lessened, even if not completely eliminated, and that less general disorganization and individual hardship would be caused by refusal at the outset to accept persons with definite clinical abnormality of the naso-pharynx as members of midwifery units. Medical examination is a recognized preliminary to the acceptance of applicants for nursing training. There seems no reason why, in the case of midwifery trainees and those applying for senior permanent positions, there should not be examination of the history and condition of the upper respiratory tract by a rhinologist, with cooperation of bacteriologist and radiologist if necessary.

Discussion.

It will have been noted that no action was taken in the outbreak of puerperal sepsis described in this paper until three cases had occurred. It is outside the scope of this paper to discuss the problem of how best to avoid such undesirable delay, but it may be remarked that almost all experienced workers stress the extreme importance of bacteriological investigation of every case of puerperal pyrexia as soon as the fever is manifest. It should be just as much a matter of routine in these cases to take a swab from the cervix or upper part of the vagina of the patient and from the throats of her contacts as it is to take swabs in a case of suspected diphtheria. If no hæmolytic streptococci are found, other causes for the pyrexia must be looked for. Incidentally, the relief from uncertainty and anxiety afforded by the knowledge that the patient's fever is not due to a streptococcal infection has only to be experienced to be realized. On the other hand, should the swabs show hæmolytic streptococci, treatment with drugs of the "Prontosil" group can promptly and confidently be used. When such a valuable but highly specific drug is somewhat expensive and sometimes difficult to obtain, there is even less justification for the omission of a preliminary bacteriological examination than before its introduction.

In addition to serving the best interests of the individual patient, the prompt investigation of any case as it arises, especially if in hospital, ensures that in the event of its being streptococcal, the risk of dissemination of these organisms, both by physical agents, such as dust, the possibility of which was demonstrated by Elizabeth White,[17] and by the creation of carriers among the contacts, will be far less than it may be after some days' delay. Had the outbreak described above been limited to the first patient (Mrs. A.), the number of professional contacts would have been 13, as opposed to the 32 contacts of the three patients. Also, apart from the greater tragedy of a second fatal case, the isolation of one patient and her contacts would have caused far less disorganization than the drastic measures necessitated by the further spread.

We did not attempt to study the epidemiology of this outbreak by Griffith's method. The work of Dora Colebrook[2] and Smith[8] on puerperal infection, and that of Okell and Elliott[18] on cross-infection in oto-rhinological wards, which follow Griffith's earlier work, have produced convincing evidence of the close relationship between throat carriers and streptococcal infections. Their work has therefore provided a strong argument in favour of attempting to lessen the incidence and spread of puerperal infection by prevention of transference of streptococci from nose and throat reservoirs. For this latter purpose, however, it is usually not necessary or even desirable to wait for the results of detailed serological differentiation by Griffith's method (which is both difficult and time-consuming), since any Group A strains encountered, irrespective of their type according to Griffith's classification, and possibly those of other groups must be regarded as potentially pathogenic. It should be noted, however, that if full identification by Griffith's or some other suitable method is not made, it is rarely possible to state with certainty that the presence of Group A or other hæmolytic streptococci in the throat of an attendant is directly associated with a patient's infection. For this reason it appears only just (unless there has been failure to report a "cold" or other non-adherence to the required technique) to emphasize, when dealing with nurses, that swabbing after the occurrence of sepsis is undertaken to prevent further spread, rather than as an attempt to assign the blame for the existing case.

Neither did we consider it necessary for our purpose to classify the streptococci which did not fall into Group A, owing to the paramount importance assigned to this group in connexion with human disease by Lancefield and her fellow pioneers. Evidence is accumulating, however, that the other groups cannot be entirely neglected. Hare[6] has already reported two cases of fatal puerperal infection from which Group B strains were isolated; Plummer[19] has identified Group C strains in two cases, and strains belonging to several groups have been isolated from cases of puerperal sepsis at the Women's Hospital, Melbourne.[20] These cases emphasize the need for caution in dealing with throat carriers of streptococci other than those belonging to Group A.

Summary and Conclusions.

1. A brief description is given of an outbreak of puerperal fever, involving three patients in a maternity hospital. The measures adopted to deal with it and those designed to prevent future similar outbreaks are outlined.

2. The value and limitations of periodic swabbing of the nose and throat of midwifery workers are discussed.

3. The bacteriological technique is described. In addition to the recognized biochemical and serological methods of classification of hæmolytic streptococci, their sensitivity to four serologically distinct types of streptococcal bacteriophage was tested; it was not found to be in complete agreement with the results of the precipitin test. The variation of sensitivity to bacteriophage among strains falling into Lancefield's Group A suggests, however, that this property might be used in epidemiological studies.

4. The incidence of throat carriers of Group A streptococci among the contacts of the three patients with puerperal fever was found to be 22%. On two subsequent occasions, when there had not been recent known contact with streptococcal infections, the incidence of Group A streptococci among similar groups of medical practitioners and nurses was 3% and 6·7% respectively.

5. Cases are described and tables given illustrative of the varying problems encountered in different individuals.

6. In persons with healthy respiratory tracts the presence of hæmolytic streptococci in throat swabs appeared to be closely associated with recent contact with streptococcal illness. In those with clinically detectable abnormality of the nasopharynx there was a greater tendency to harbour hæmolytic streptococci irrespective of recent contact. It is therefore suggested that exclusion from midwifery units of persons with such abnormality, by means of preliminary medical examination, would be a possible way of reducing the risk of infection of patients and the disorganization attendant on their withdrawal during a period of midwifery work.

Acknowledgements.

We wish to thank the medical men and women and hospital authorities whose cooperation made this work possible. We greatly appreciate the interest shown throughout by Dr. F. M. Burnet, assistant director of the Walter and Eliza Hall Research Institute, and to thank him for much helpful criticism of this paper, given in the absence of the director, Dr. C. H. Kellaway.

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FURTHER OBSERVATIONS ON DELAYED TESTIS.[1]

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In introducing this subject I feel that, at the outset, I should make some explanation of the use of the word "further" in my title. Many of you may be excused for not knowing that fifteen years ago The Medical Journal of Australia published a paper on "Delayed Testis" in which were summarized observations on the incidence, progress and prognosis of this congenital condition, as revealed in the routine annual medical inspection of the boys at Scotch College, Melbourne. The present paper is offered, with fifteen years of added experience and written records to draw upon, largely in the nature of a control or review of the original one. It is fitting here to acknowledge gratefully the courtesy of the head master, Mr. Colin Gilray, in making available the record cards for investigation, and also the sympathetic assistance of Dr. Euan Littlejohn, the college medical officer, whom I was assisting in the medical inspections.

I should like to make it perfectly clear that this contribution is, in the main, statistical, and does not set out to lay down any specific directions as to the form of treatment to be adopted. Suggestions there may be, but nothing more. The clinical side of the question may well be left to abler and more experienced workers in the field. One recognizes, of course, also, the dangers that beset the path of the would-be translator of figures into facts. It is hoped that the present observations will be of some value in throwing light more particularly on the progress of these dilatory testes as their owners grow up from small boys to puberty and adolescence.

The investigation is derived from an analysis of the physical record cards of 3,197 schoolboys, whose ages ranged from five to eighteen years. The boys are examined carefully in their first year, the result of the examination being recorded in detail on their cards. Each succeeding year of the boy's stay at school he is re-examined and any abnormal feature noted previously is carefully checked over. When this system was initiated in 1919 special attention was devoted to the inguinal regions to determine the existence or not of herniæ—potential or actual. The late Dr. Hobill Cole drew my attention to the comparative frequency of delayed descent of the

[1] Read at the fifth session of the Australasian Medical Congress (British Medical Association), August, 1937.

testes, either unilateral or bilateral, and the scrotal and inguinal regions have been closely inspected and results recorded ever since then.

Standard Adopted.

The effect of cold air, nervousness and handling of the scrotum in the course of the examination, in causing the cremasteric retreat of the testis to the shelter of the inguinal canal, necessitated the adoption of some standard by which the real delayed testes could be recognized and the pseudo ones discarded. "Any testis which, being found absent from the scrotum, could not be located in the inguinal canal or external ring, drawn down or coaxed down and induced to stay in the scrotum without tension" was regarded as being a genuine delayed testis. This may be open to criticism, but I am quite sure that, with experience, one is able to distinguish the false from the true in most cases and that, with patience, the wandering or cremasterically reflected testis can be dislodged from its hiding place and induced into the scrotum. Among the small boys in the Junior School between five and seven years of age, if the weather is cold, the examination of the scrota is deferred for twelve months or more, and in the present year's examination we must have dealt with many in this way.

Let us turn now to an analysis of the figures as set out in Table I. For purposes of comparison the figures for the 1922 investigation are set out in parallel with the present figures.

TABLE I.

Class.	Number Examined.		Delayed Testes.		Percentage.	
	1922.	1937.	1922.	1937.	1922.	1937.
1, Boys 15 years and over ..	590	1,066	1	12	0·16	1·1
2, Boys under 15 years	1,255	2,131	123	192	9·8	9·0
Totals ..	1,845	3,197	124	204	6·7	6·08

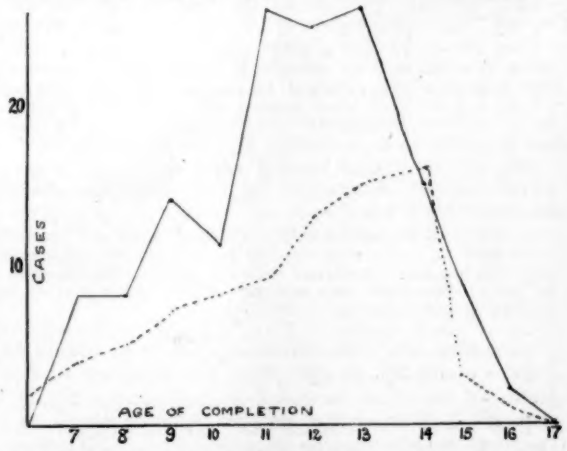
From the cards of 3,197 boys, 12 or 0·37% of the total were discovered to have delayed testes at the age of fifteen or over. By way of contrast, 192 boys under the age of fifteen were found to exhibit delayed descent, or 6% of the total number. The actual incidence of delayed testis in the 3,197 boys works out at 6·08%. The proportion of boys under fifteen to those over fifteen in the school is roughly 2 to 1, and the numbers have been divided into Class 1 and Class 2 in order to separate in an approximate manner the boys who have attained puberty from those who have not. There is a very striking difference in the percentages revealed. Of the boys under fifteen, 9% exhibit delayed testis, whereas those fifteen and over show only 1·1% so affected. Something occurs at the fourteenth or fifteenth year to hurry on the lagging testis with the result that a 9% frequency is converted into a 1·1% comparative scarcity. It is almost certain that developmental changes and general hurrying up of physical growth, associated with the onset of puberty, are the factors at work. At this point a suggestion arises with regard to the treatment of these cases. Should we not ask ourselves first: "What can puberty do for this boy to bring him back to normal before proceeding to institute surgical treatment?" The translation of figures into facts in this case, even if literal, should make common sense reading. We can at least say with some justification that the majority of delayed testes appear to attain their normal scrotal position before or about the age of puberty.

Relationship of Completed Descent to the Age of Completion.

We have seen that only twelve boys out of 3,197 showed incomplete descent of the testis after the age of fifteen. It is interesting to note that in neither series of investigations has there been one instance of bilateral delayed descent after the age of fifteen in a total of 5,000 boys. This should have an important bearing on the suggestion of the risk of sterility supervening if these boys are not surgically treated, as one healthy testis normally placed should be sufficient to guard against this contingency. Of the twelve unilateral cases in this series, in nine cases the left testis was the laggard.

Of the remaining 192 cases in boys under the age of fifteen, 133 testes were noted in the follow-up years as completing their descent before the age of fifteen, eight more were completed at the age of fifteen, and two even at 16, so that 143, or 74%, descended spontaneously while under observation.

A reference to the graph shows that in these 143 cases, eight testes completed descent at seven years, the numbers rising to 26 at eleven and thirteen years, and then falling to fifteen at fourteen years, with a steep slope down to zero at seventeen years.



Graph showing number of cases and age of completion. The interrupted line indicates the observations made in 1922; the continuous line indicates those made in 1937.

The remaining 49 boys under the age of fifteen are still under observation, or have left the school, and no doubt in the majority of these the testes will attain full descent before or at puberty.

Side Affected.

Of the cases 55% were noted as being bilateral, 30% were right-sided and 15% left-sided. Six pairs of brothers are included in the list.

Relationship of Delayed Testis to Physical Type of Boy.

The investigation showed very little evidence of any constant relationship between delayed descent and poor physical type of boy. Most of the boys conformed fairly well to average physical standards, although in a few instances boys were noted as being short in stature, with infantile genitalia, some even approaching the *dystrophia adiposo-genitalis* type. With the onset of puberty these boys seemed to improve quite rapidly.

Relationship to Inguinal Hernia.

The relationship to inguinal hernia was discussed in detail in the 1922 series. The alleged increased liability to inguinal hernia as a sequela to late descent of the testis has been adduced in the past as a reason for operating on these boys early, say, between the ages of six and nine. The persistence of a patent funicular process after late descent, as recorded by the late Mr. Hamilton Russell, would appear to support this theory, but the figures as shown in the present series confirm fully the observations made in 1922. Only four inguinal herniæ were discovered in the 204 cases of delayed testis, and they are here presented in detail:

Case I.—R.P., aged seven years, was noted in 1924 to have a left delayed testis. In 1925 the left testis was in the canal—the boy had a potential hernia. In 1926 he had a left inguinal hernia, the testis was still in the canal. In 1927 the hernia was larger. His condition was reported for the third time to his parents as urgent. In 1928 the hernia had been operated upon. The testis was in the scrotum; some swelling was present and the site was slightly painful. In 1929 the boy was normal. In 1933 he was finally reviewed as quite normal.

Case II.—H.G., aged eight years, in 1929 had a right testis delayed and an associated right inguinal hernia. The condition was reported to the parents and nothing was done until 1934, when examination showed that herniotomy had been performed and the testis in the scrotum had been grafted to the thigh. The boy left school.

Case III.—P.R., aged thirteen years, was found to have a right herniotomy scar and the right testis was absent. Its fate is unknown.

Case IV.—K.F., aged fourteen years, in 1929 had his left testis high up in the inguinal canal. In 1930 the testis was still delayed and associated inguinal hernia was reported. In 1931 herniotomy and removal of the testis had been performed three months previously.

Assuming that the normal incidence of hernia in boys is about 2% to 2·5%, and the incidence in this series, of four cases in 204, is slightly under 2%, the sequential relationship between delayed testis and inguinal hernia may be regarded as very doubtful indeed.

Suggestions as to Treatment.

My translation of figures into facts may be regarded as somewhat free in this connexion, but certain suggestions arise from the analysis which at least demand consideration.

1. Except in certain cases in which there is some associated surgical condition, such as hernia, hydrocele, or torsion of testis *et cetera*, surgical intervention should be withheld at least in the first ten years.
2. If, after that age, there is evidence of persistent delay associated with under-development of the genitalia, the exhibition of gonadotropic stimulus with "Antuitrin S" or similar preparation might be considered advisable.
3. If with the commencing appearance of the secondary sexual characteristics (development of pubic hair *et cetera*) the testis still shows no sign of descent, then surgical intervention is definitely indicated.

Conclusions.

In 1922 I arrived at the following conclusions:

1. That delayed testis is a common condition in boys under the age of puberty, being relatively nearly three times as common as inguinal hernia.
2. That after the age of puberty the condition is relatively rare.
3. That the normal hurrying up of developmental processes associated with the onset of puberty plays a part in hastening the completion of descent of the delayed testis.
4. That the sequential relationship between delayed testis and inguinal hernia is not proven and the weight of evidence is against it.
5. That the condition displays a definite and constant tendency to spontaneous cure and that routine surgical interference before commencement of puberty is contraindicated.

In 1937, with more material to work on and more experience in the work, I find no reason to alter my conclusions.

Finally, I should like, as a seeker after the truth, to issue in the friendliest possible manner a challenge. Doubts have been cast by surgical colleagues on the possibility of the incidence of delayed testis being nearly as high as I have stated. These doubts are based mainly on clinical impressions and not on the solid evidence of systematic examination in the mass and recording of results obtained. Clinical impressions in retrospect may be even more misleading than statistics and are relatively of little value compared with clinical observations which are immediately recorded as they are made. I would suggest, therefore, that until an investigation is made along the lines of the present one (and school medical inspections are almost ideal for the purpose) and results are recorded which substantially disprove the figures I have given, I have every right to ask that they be accepted as a reasonably accurate statement of the case.

Addendum.

Review of Gonadotropic Treatment.

Treatment along the gonadotropic lines is reviewed very well and comprehensively in the "Medical Annual", 1937.

In 1922 Evans and Long demonstrated the existence of a gonad-stimulating hormone in the anterior lobe of the pituitary gland, controlling the activities of the ovaries and testes.

In 1928 Aschheim and Zondek isolated a gonadotropic principle from pregnancy urine.

In 1932 Engle applied this principle in treating immature monkeys in which the testes had not yet become scrotal, with successful induction of descent; and since then much work has been done along these lines.

In 1933-1934 Burrows demonstrated the activity of œstrin on mice in relaxing the musculature and so predisposing to scrotal hernia. He used bi-weekly applications of œstrin to the interscapular region and produced scrotal hernia. He suggested that the œstrogenic hormone inhibited the maturation and descent of the testis, and acted in balance to the gonadotropic hormone which stimulated these. The œstrogenic substance predominated when the testis was intraabdominal, and the gonadotropic hormone assumed control in the last month or two of fœtal life.

In 1935 Spence and Scowen, working on 33 patients ranging in age from four to twenty-six years, applied treatment with 500 rat units of "Pregnyl" twice weekly, and claimed results which were by no means uniformly successful.

In 1935 Wangensteen reported optimistically on the recovery of spermatogenic function in testes that had had orchidopexy performed, and his views were supported by Pearse Williams.[1]

In 1936 McLellan assessed his results under three groups as follows:

Group.	General Physical Development.	Development of Testes.
I	Normal.	Normal.
II	Subnormal.	Subnormal.
III	Normal.	Subnormal.

Group I showed little if any response. Group II showed uniform improvement (not necessarily descent in every case, but genital development at least). Group III showed some benefit but nothing very definitely encouraging. His results would appear to indicate that the gonadotropic hormone acts best in the subnormally developed tissues which appear to require it most.

In 1937 T. W. Mimpriss (*The Lancet*, February 27, 1937) reported observations on 20 patients treated with "Pregnyl", 500 rat units, twice weekly.

1. In 19 cases hypertrophy of the external genitalia resulted.

2. In six cases descent was achieved, five of these cases were bilateral. The ages of the boys were: six, nine, ten eleven, eleven, and thirteen. Mimpriss's conclusions were that no results need be expected in boys whose genitalia were well developed, and that treatment was mainly of use in bilateral cases with subnormal development. The dangers of premature maturity constituted a serious objection to the treatment, which certainly should not be instituted before the age of nine.

Reginald Miller's conclusions, with which I feel impelled, in the main, to agree, are as follows:

1. The true cryptorchids where the testes are in the abdomen show very little prospect of spontaneous descent. They should be watched carefully until after the ninth year. Hormone treatment should be instituted as puberty begins to develop, and, if there is still no result when puberty is established, surgery should be invoked.

2. Uncomplicated canal-dwelling testes may be, in most cases, expected to descend spontaneously. As before, if there is no descent with the advent of puberty, give hormone injections. In any case do not delay after the age of 14.

Cases complicated by hernia or hydrocele should have the complications dealt with surgically, and the testes brought down if possible.

[1] One boy in the present series was noted as showing descent following herniotomy without orchidopexy.

TRAUMATIC RUPTURE OF THE URETHRA AND ITS TREATMENT[1].

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THE material which forms the basis of this paper was obtained by a study of the 33 cases of traumatic rupture of the urethra admitted to the Royal Melbourne Hospital during the last ten years. With few exceptions, attempts to follow up the patients for any length of time were unsuccessful. These patients had ruptures of all grades of severity, from partial to complete. It is obvious, both on general lines and from a study of the histories, that the prognosis in the patients with partial rupture is infinitely better than in those with a complete rupture; this will be referred to later.

Classification of Lesions.

For the purposes of treatment, a useful working classification is to divide these cases into three groups according to the nature of the accident which has produced the injury. So that we have: (i) rupture due to the passage of instruments, one case; (ii) rupture due to direct injury to the perineum, for example, a blow in the perineum or a fall on to the perineum, 20 cases; (iii) rupture due to a fractured pelvis produced either by a crushing injury or in street accidents, or by a similar type of injury in which the patient is either struck in the side or thrown violently to the ground, 12 cases. This classification is essentially practical since the types are easily recognized on the history, which in spite of the modern diagnostic aids still remains an important factor in reaching a correct diagnosis. In addition this classification is of value in that the treatment accorded to the three types varies somewhat in detail.

Group I.

The solitary case occurring in the first group was due to the passage of a cystoscope. It is merely mentioned to complete the series and to serve as a warning in these days of more or less indiscriminate cystoscopy. The patient runs a risk of a subsequent stricture. In this class should be considered the more frequent injuries due to inexpert passage of sounds and catheters.

Group II.

The second group (that in which there is direct injury to the perineum) is the most varied in its manifestations and in the lesions produced, and in the more severe forms is apparently the most liable to give rise to bad late results. In two cases in this series the injury was so severe that a fracture of the pelvis was produced in addition to rupture of the urethra. For the purposes of discussing treatment one of these can be excluded, as the patient died from the severity of his injuries very shortly

[1] Read at the fifth session of the Australasian Medical Congress (British Medical Association), August, 1937.

after admission to hospital. The other case in this group that was associated with fracture of the pelvis was that of a woman who was riding pillion on a motor cycle which was involved in a collision. Since rupture of the urethra is very rare in women, it is worth giving the history of this patient in some detail, particularly since it also illustrates some of the principles of treatment.

On admission to hospital she was complaining of severe pain over the pubis, the pain being made worse by movement. The bladder extended half way to the umbilicus. Vaginal examination disclosed lacerations in the anterior wall of the vagina, including the urethra. A bone end, thought to be the anterior ramus of the left pubis, was palpable in the wound. Immediately after this the patient was reported to have passed urine naturally. She was then given a general anæsthetic, when a separation of the pubic bones was found, with laceration of the vulva and urethra. A self-retaining catheter was inserted into the bladder and fixed with a silkworm gut suture and a bladder wash-out given. The catheter was removed on the fifth day. The next day it was noticed that vaginal bleeding had occurred and that some of the areas of laceration were sloughing. The bleeding was more severe on the following day when she was given a blood transfusion and a collection of pus was opened. From then on she gradually improved and was discharged to the Caulfield Convalescent Hospital, nine weeks after her admission to the Royal Melbourne Hospital. At this stage she had fair control of micturition when resting but practically no control when moving about. She was readmitted to hospital four months later in the care of the gynæcologist, because of her complaint of constant dribbling when standing. At this time the external meatus was absent and the urethra was practically non-existent, and gave the impression that it had been split along its floor. A plastic operation was then performed, what remained of the urethra being freed from the bone and dissected out. The patient made a good recovery from this operation, and she had better control of micturition. She was readmitted to hospital one year later, because she still suffered from dribbling of urine after walking for more than twenty minutes. On examination she was found to have a small urethro-vaginal fistula and a cystocele. The fistula and cystocele were repaired. After this she made a good recovery and since her last operation has not complained of incontinence.

In reviewing this case the question arises as to whether suprapubic drainage performed immediately might have shortened the patient's stay in hospital on the first occasion by lessening the risk which she undoubtedly ran from infection of the vaginal lacerations. Of course, an attempt to make an immediate suprapubic incision for drainage involved a risk of her passing urine while the anæsthetic was being administered. It does seem as though this would have been the better course, certainly better than allowing her to pass urine naturally when it was known that she had extensive lacerations to the vagina. The reason for this preference is that it was the infection of these lacerations which caused the sloughing and secondary hæmorrhage, for which she was given a transfusion on the seventh day. Suprapubic aspiration might in her case have been worth considering while she was being prepared for operation.

As regards the later management of this case, one cannot but commend it. The patient was given some months to recover from the initial injury before any attempt was made to perform a plastic operation. Then, after the first operation, she was given a full year before any attempt was made to improve the result. Such restraint is not always exercised, but it undoubtedly has everything to commend it, and in this case received its reward in the excellent functional result that was finally attained.

In the other 18 cases of this group, which were not associated with fracture of the pelvis and which constitute the common type of ruptured urethra, space does not allow the description of individual cases. The main symptoms were (i) hæmorrhage from the urethra, which was present in all except two, (ii) inability to pass urine and (iii), pain and swelling in the perineum. In eight of these cases the only treatment was the passage of a catheter, which was tied in in every case except one, in which the injury was to the penile urethra. As there was no difficulty in introducing the catheter in these cases it is assumed that they were all examples of incomplete rupture. As far as can be ascertained by a study of the out-patient records, none of these patients had any trouble later. Some of the other ten cases will be described, but as they do not divide up into very definite groups they will be considered in chronological order, since this will help to bring out the increasing tendency to use suprapubic drainage as the first step in any case presenting difficulty, which is without doubt the correct procedure to adopt.

Of these ten patients, one died soon after operation, and one died nine months after his injury from extravasation following the closure of a perineal fistula. Three patients had persistent strictures, for which they are still receiving treatment; so that in this group of those requiring operation, half have died or are suffering from serious complications.

The first patient was admitted to hospital in April, 1927, with the typical history of falling astride a wooden beam. He had more evidence of injury to the perineum than is often the case. He was operated on immediately, a median perineal incision being made, and the urethra was found ruptured in the region of the bulb. A rubber catheter was passed into the bladder with much difficulty, and brought out through the perineal wound. The patient had a perineal fistula for eight months, and still, ten years later, attends the urological out-patient department with a stricture which admits only a number 14 French gum elastic bougie. He illustrates, in addition to the disadvantages of perineal drainage, the greater risk of fistula in a thin individual, a point which has been referred to in the literature.

Case II is remarkable in that the patient was the only one treated by perineal incision and perineal drainage who did not develop a fistula. The rupture in his case was not complete.

In Case III a suprapubic incision was made to allow retrograde catheterization as an aid to finding the proximal end of the urethra. No attempt was made to pass a catheter through the penis into the bladder, but perineal drainage was instituted. This was partly because generalized fibrocystic disease of the patient's skeletal system with deformity on his left hip added to the difficulties of the operation. He had a perineal fistula which functioned for eight months and then closed, and his suprapubic wound opened. He was readmitted to hospital with signs of extravasation in his right thigh, and died four days later.

This patient illustrates another point, in that when attempts were made in the casualty room to catheterize his bladder it was thought that the catheter was in the bladder, although no urine was obtained. To test this, fluid was injected up the catheter, but the only result was the causing of pain and swelling in the patient's left ischio-rectal fossa. Such injection of lotion cannot be too strongly condemned, for even if the catheter is in the bladder it probably means that the bladder is ruptured. We have obvious demonstration of the risks in this case, in which it was plainly evident that fluid was being forced through the ruptured urethra. This case serves also to show the inadvisability of washing out the urethra as a preliminary to catheterization as advocated by some writers, since if this is done fluid must be forced through the rupture.

Case IV illustrates two points of interest. The patient was transferred from a mental asylum, after a kick in the perineum received two days previously. The first point is that he passed urine naturally for twenty-four hours and then suffered from retention. This is a well recognized feature, but his is the only case in our series illustrating it. The second point of interest is that his bladder was catheterized easily on his admission to hospital and a catheter was tied in. There was some difficulty in keeping the catheter in, and on the tenth day the catheter came out and could not be replaced; there was obvious extravasation of urine into the perineum. Operation revealed a complete rupture of the urethra. Although the local condition improved after operation, the patient died four days later. It is an interesting speculation as to whether the rupture was complete from the start.

From this stage on in this series a suprapubic incision was much more freely used. It was used, too, as the first step in treatment rather than as a way out of a difficulty, which was the way it was regarded up to this stage. In none of the cases after this was perineal drainage used. It would appear that in some of the earlier operations in this series the operators were influenced by the teaching of Hamilton Russell in advocating perineal drainage after excision of strictures. Although in Russell's operation perineal drainage is a sound procedure, in these cases it is not, as the catheter is brought out through damaged tissues. It is better to have a catheter along the whole length of the urethra to act as a splint, and if drainage above the repaired urethra is required then it should be provided by suprapubic drainage of the bladder. Mr. Russell's idea would be carried out in these cases by uniting the roof of the damaged urethra and leaving the remainder of the wound unsutured, drainage then being provided by a suprapubic tube in the bladder. This method was not tried in this series, but was suggested as a possibility, with due acknowledgement to Mr. Hamilton Russell, by Sir William J. de C. Wheeler, in opening a discussion on this subject at the Royal Society of Medicine in January, 1929.

Case V was the only one in this group in which the rupture occurred in the membranous urethra. The patient was one of the two who did not suffer from hæmorrhage from the urethra. He was operated on by combined suprapubic and perineal incisions with repair of the urethra over a catheter. He later developed an impassable stricture with perineal fistula; the stricture was excised later by the urologist. He is still attending the urological department, but will take a 21/23 French metal sound quite easily at the present time, and has no urinary disability. A urethrogram reveals narrowing at the junction of the membranous and bulbous urethra.

Case VI is the only example in this series of an incomplete lesion giving rise to great difficulty in treatment. The patient was treated by what is coming to be recognized as the standard method, that is, by suprapubic drainage as the first step, followed by exploration of his urethra. It was found possible to manipulate a catheter through the urethra, using a retrograded sound as a guide. The catheter was then cut down on in the perineum and the urethra was found to be pulped for about 3·75 centimetres (one and a half inches) but not actually severed. He had trouble for some months with repeated hæmorrhages and a persistent swinging temperature, and was discharged from hospital with permanent suprapubic drainage. Some months later he was transferred to the care of the urologist, who performed an internal urethrotomy, since his obstruction at this stage was apparently of a diaphragm nature. Since then he has gone straight ahead and now takes a number 20 French bougie easily without bleeding or discomfort and his suprapubic wound is firmly closed.

I had intended to discuss the individual cases more fully, but representative cases have been selected and some of the difficulties outlined. One point that I intended to make is the increasing dependence on suprapubic drainage. Kidd, in the discussion referred to above, stated quite definitely that the most important step in treatment in any case giving difficulty was suprapubic drainage, and he also affirmed that if that was established, it did not matter very much what was done to the urethra.

Group III.

In Group III there were 12 cases, and in passing it is interesting to note that 11 of them occurred in the last five-year period; seven of these were due to street accidents, so that the inference is that this is a lesion which may easily become more common. Because of the severity of the injury a greater proportion of these patients will succumb within a short period. In this group of 12 there were five deaths; of these, four occurred within a short time of the injury, and one patient died from apparent renal failure three months after his injury, although he had apparently recovered and there was no evidence of urinary obstruction. In spite of the variations in the injuries there was a remarkable unanimity in the treatment, which in all patients coming to operation was primary suprapubic drainage. This is without doubt the proper line of treatment, as the rupture is almost always intrapelvic; in other words, the rupture is above the urogenital diaphragm and there may be an associated rupture of the bladder.

Sir William Wheeler quotes a patient who was thought to have a ruptured bladder, but a catheter withdrew clear urine. The patient, however, grew steadily worse and was then operated on; a ruptured bladder was discovered, and it was shown that the catheter had gone through the rent in the bladder and drained clear urine from the peritoneal cavity. One patient in this series had a rupture of the bladder in addition to a rupture of the urethra, and it is obvious that this could not be determined except by operation.

The usual site of the injury is somewhere between the attachment of the urethra to the urogenital diaphragm and the base of the bladder. Quite commonly the bladder and prostate gland are torn off the urogenital diaphragm and displaced backwards and upwards into the abdominal cavity, causing a wide separation between the divided ends of the urethra. The suprapubic approach is the best in this type of injury, since the distended bladder can be emptied and the blood clot removed from the cave of Retzius. It is essential that a catheter be introduced through the whole length of the urethra to act as a splint, keeping the bladder and prostate in contact with the distal urethra. Many devices have been described for getting a catheter through. Actually there is usually no difficulty in getting a catheter passed through the penis to present above the urogenital diaphragm, and it can often be grasped and pulled into the bladder by curved forceps passed down through the internal meatus. If this manœuvre fails, a catheter can be passed down from the bladder and then joined to the end of the one previously passed up the urethra, which is then drawn up into the bladder. A. Simpson Smith in *The British Journal of Surgery*, October, 1936, described a method for providing traction in these cases by the use of a rubber collar round the catheter in the urethra; this collar pulls on the bladder base round the internal meatus. Such traction does not seem to be necessary; the main thing is to get the catheter through at the time of operation and get the alignment of the urethra.

In other cases, in which separation has not occurred, after the bladder is opened the position can be reviewed, and attempts can be made to pass a catheter either by the retrograde method or along the urethra. If a catheter can be got through it should be tied in, and nothing further should be done, the patient being left with drainage through the catheter and suprapubic drainage. If a catheter cannot be got through, a sound should be passed from each end of the urethra; their ends should then be cut down on, and the catheter manipulated along the urethra to the bladder.

In this group there was no patient with a rupture of the bulbous urethra, which contrasts with the frequency of this lesion in the other group, in which there was only one rupture of the membranous urethra. Again, in contrast with the other group, only one patient's bladder was successfully catheterized; he was fortunate, as he had a fracture of the femur in addition to his fractured pelvis, and was put up in a Whitman plaster almost immediately, a procedure that would have made nursing very difficult with suprapubic drainage.

This group of patients with fractured pelvis has been dismissed rapidly, not because they are not considered important, but because the line of treatment seems so well defined. All who survived the initial period did well, except one who had extensive pelvic injuries and in whom the suprapubic wound could not be made to close. He had collections of pus opened in the pelvis and the perineum. He was in the Royal Melbourne Hospital for nearly fifteen months before being transferred to a country hospital. He was a man of thirty-eight years of age with extremely severe injuries. A permanent suprapubic tube should be manageable, but in some of these patients transplantation of the ureters is a way out of a difficulty. This would not be applicable in this patient, as he has had infection of both kidneys with calculi during his stay in hospital.

Treatment.

Since a large proportion of patients (8 out of 18) suffering from hæmorrhage and inability to pass urine after direct injury to the perineum (Group II of our classification) responded to the tying-in of a catheter without any other treatment, it is obvious that this should be tried as a first step. This attempt at catheterization should, however, be undertaken only in the operating theatre with the strictest of aseptic precautions. In no circumstances whatever should catheterization be attempted in the casualty room. If there is urgent need to empty the bladder, this can best be done by suprapubic puncture and aspiration, a fine trochar and cannula being used; but such a procedure would hardly be necessary in hospital practice. It is the method to be adopted if there is going to be any delay in getting the patient to hospital. For this attempt at catheterization it is not necessary to give the patient an anæsthetic, but he should be prepared as for operation. This preparation should include the shaving of the perineum and the preparation of the skin of the perineum and lower part of the abdomen. If the attempt at catheterization fails, then the patient should be anæsthetized, and the surgeon should go on and provide suprapubic drainage. It cannot be too much emphasized that this attempt at catheterization should be made only in those cases in which the injury is due to direct violence to the perineum. In those cases in which the rupture is due to a crush or fall, with a fracture of the pelvis, no attempt should be made to pass a catheter, but suprapubic incision with drainage of the bladder should be the first step. Occasionally this may turn out to have been unnecessary; but much more often a ruptured bladder or a bladder separated from the urogenital diaphragm will be found, in which no other procedure but suprapubic incision would enable the operator to deal with the lesion. In addition, cases are on record in which the passage of a catheter in this type of lesion has been misleading, in that the catheter has passed through a tear in the bladder and drained urine from the peritoneal cavity, thus producing a false sense of security in the surgeon. Therefore, because it is almost invariably futile and when not futile may be misleading, catheterization in cases associated with fracture of the pelvis due to crushes and violent blows on the side, must be condemned.

In this type of injury (Group III of our classification), since the damage is almost always intrapelvic, the actual lesion can be remedied

through the suprapubic incision at the time of this first operation, so that as a rule no further operative procedure is required. In the other type of injury (Group II of our classification), after the bladder has been drained the question arises as to how the urethra should be dealt with. In these cases we know the lesion is more commonly below the urogenital diaphragm, hence another incision in the perineum may be required to deal with the lesion.

After the bladder has been drained the following three courses are open. Firstly, it may be decided to do nothing further and to leave the treatment of the injured urethra till a later date. This procedure was not adopted in the series described, but has been used by Wheeler and is favoured by Marion. It does seem that it would be worth considering if there were much swelling and injury to the perineum, and particularly when much extravasation occurred, since incisions could be made in the extravasated area for drainage and then later, when the swelling had subsided, the urethra could be dealt with under much more favourable conditions. Secondly, the urethra might be explored by means of a rubber catheter, either from above or below, or by combined methods. This would usually be tried, and if a catheter could be got through, it should be tied in, and nothing further should be done at this stage. Thirdly, it might be decided to repair the urethra immediately. In this case it is a great advantage to have the suprapubic opening so that a sound can be passed down from the bladder as a guide to finding the proximal end of the urethra, which is always the most difficult step in the operation. In this way time is saved and damage to the perineum is reduced to a minimum. Although repair was carried out in some cases in this series, in none does it seem to have been as thorough and as methodical as that of Wheeler. This covers the immediate treatment of these patients; strictures occurring as a late result are treated on general lines.

In preparing this paper I experienced the same difficulties as have been emphasized by other writers. Although most of the patients in this series who presented any difficulty in treatment were referred sooner or later to the urological department, on admission they were placed under the general surgeon. This series of 33 patients, extending over ten years, were attended by 13 surgeons, so that over that period each surgeon would see between two and three cases. It is apparent, therefore, that no one surgeon obtained experience of any value in the treatment of these patients. Even under these adverse conditions it is possible to trace a gradual evolution in the methods of treatment, but it is obvious that with such limited experience it is not possible for a surgeon to develop a sound judgement as to the proper line of treatment, or to develop an adequate technique for dealing with the more serious lesions. This applies particularly to the cases of direct injury to the perineum, in which sound judgement in deciding how much or what should be done after suprapubic drainage has been provided may make much difference to the future health and comfort of the patient. This knowledge could be obtained only if it were agreed to place all these patients under one surgeon, or one group or team. We must consider the question of service to the community, and there can be no doubt that the community would be better served by some such arrangement. If the figures in this series are taken as a guide, it will be seen that each general surgeon might lose two or three cases over a period of ten years; and as they are probably the only cases he will ever see, it does not seem too much to ask that, for the sake of more certain and efficient treatment, he should transfer them to the care of one group, who would then be in a position to see a greater number of such cases.

Summary.

1. A study has been made of 33 cases of rupture of the urethra occurring at the Royal Melbourne Hospital in the last ten years. This number includes all grades of rupture, from partial to complete. Patients with partial ruptures almost invariably do well, irrespective of the treatment. They cannot be recognized till treatment is initiated.

2. A practical classification into three groups is suggested: Group I, consisting of those whose ruptures are due to instrumentation; Group II, consisting of those whose ruptures are due to direct injury to the perineum (usually ruptures of the bulbous urethra); and Group III, consisting of those whose ruptures are due to crushes and to violent blows on the side, and are associated with a fracture of the pelvis, the ruptures being usually intrapelvic in position.

3. Treatment in all three groups has been discussed.

4. It is suggested that, owing to the rarity of the complete lesion, all these patients should be put under one surgeon or one group, who would then be in a position to develop a sound technique based on an informed judgement.

THE TURBULENT DR. LHOTZKY.

By JOHN MACPHERSON, M.A., B.Sc., M.B., Ch.M. (Sydney), *Sydney*.

JOHN LHOTZKY (or Lhotsky) was an expatriated Pole. He held the degree of M.D., but does not appear to have practised medicine in Australia. He was essentially a scientist and naturalist, and was "F.R.Bot.S." of Bavaria. Lhotzky was obviously a man of education, although his spelling at times was distinctly unusual. He was particularly prone to the use of Latin to give point to his remarks. When he came to New South Wales he apparently expected to receive some official appointment, and, failing in this, he was disillusioned and embittered.

He lost no opportunity of engaging in invective against the authorities.

Lhotzky appears first in 1832, lecturing on various natural history subjects. He was keenly interested in mineralogy and proposed that an association should be formed to search for minerals. It would seem as if *The New South Wales Magazine* was begun by Lhotzky. In its second number he wrote on Australian zoology, and in connexion with the account of a seal at "Coodjee Bay" he explains what he would have done had he been appointed colonial zoologist or keeper of the Colonial Museum. Lhotzky's lectures at Hart's Buildings in Pitt Street, on mineralogy, botany and zoology, in 1833, were under vice-regal patronage, and in a subleader in *The Sydney Gazette* he was advocated as the one to fill the vacant post in the Colonial Museum. This post had remained vacant since the death of the previous holder two or three years before. The salary for the position was £150 *per annum*. Provision had been made for this amount ever since the death of the previous occupant of the position, but it had not been expended. Later, in further reference to the matter, one "T.D." writes that he is surprised that a paper with the title of *Monitor* should style it a gew-gaw, or that Mr. Wentworth's education should call it a trifle. "T.D." is emphatic in his praise of Lhotzky, and trenchantly criticizes the paltry salary attaching to the "Botanic-Cabbage-Garden which elevates our thoughts from Nature's works to Nature's God". He adds:

> It is a disgrace to this prosperous Colony, which is so rich in the animal and vegetable kingdom, as well as in the mineralogical, that we cannot say to the stranger visiting our shores: "Come to see our Museum of curiosities, which are almost peculiar to our extensive continent."

Lhotzky, in Elizabeth Street, had an excellent display of scientific objects, which evoked great admiration, and in February of 1833 there was an interesting sale of a collection of 8,000 specimens of plants, insects, zoophytes, minerals, tympanum bones of a whale, Brazil snakes, mosses, the various woods of the colony, lizards, fishes and other curiosities, the collection of Dr. Lhotzky. Captains of ships and persons proceeding to Europe were specially notified. Lhotzky was truly an indefatigable collector. We shall see later that financial embarrassment was ever a spectre before him; hence the sales, of which a further one was held in November of that year.

We read also that in connexion with his lectures on geology, mineralogy, botany and zoology his subscription list promised well, and that amongst the respectable names entered on the lists were those of Major Mitchell, Dr. Lang and Dr. Bland. Unfortunately, however, the lectures were not a success, even when illustrated by specimens. *The Sydney Gazette* was evidently a little confused when it stated that:

> Unlike most other lecturers, Dr. Lhotzky addresses his audience almost wholly *viva voce*—merely referring occasionally to notes.

It stated also that "the auditory was not so numerous as we hoped it would be".

Expedition to the Australian Alps.

In January, February and March, 1834, Lhotzky made his well-known journey from Sydney to the Australian Alps. This expedition yielded a vast amount of valuable zoological and other scientific information, but the narrative was plentifully interspersed with pungent diatribes against the authorities. Unfortunately Lhotzky's efforts gained him no advancement, as one would expect from his writings. On his return he announced his intention of publishing his journal in about twenty sheets, to be issued fortnightly. It was pointed out that this was the first work of the kind ever published in Australia, and the author expressed the hope that a discriminating public would honour him with their kind support. The expedition was undertaken by Lhotzky's "own private and very circumscribed means". Again success eluded the doctor, and only seven and a half sheets were printed in 1835. The full title of the work was "A Journey from Sydney to the Australian Alps, undertaken in the months of January, February and March 1834. Being an account of the geographical and natural relation of the country traversed, its aborigines, &c., together with some general information respecting the Colony of New South Wales." T. Iredale, of the Australian Museum, has well termed the tract "Lhotzky's Lament". He makes the following quotations from the work in *The Australian Zoologist*. In connexion with ants' nests Lhotzky states:

> I possess very fine specimens of such cellular work, but although I offered to our Colonial Government my collections, composed of all sorts of Australian curiosities, for sale, I could not get even an answer to my applications. These very curious objects are therefore now scattered about over many of the Museums of Europe, when they could, in a great part, have been united (for a comparatively trifling sum) in our Colonial Museum.

Further on, in a geological connexion, he states that:

> In my present circumstances my time is so taken up by minor business that it was impossible in my journey, and so it is now in my study, to pay to such geological occurrences the minute attention necessary. My minerals I am about to send to the Geological Society, London, and Wernerian, Edinburgh.

Again:

> The only gentleman who could render me any assistance respecting insects (*non omnia possumus omnes*) . . . is so entirely engaged in the sphere of high and official life (which, under present circumstances, seems to be incompatible with a scientific one) that it would be out of the question to request his literary assistance and co-operation. However, we will give some Entymological [*sic*] remarks in the course of this work and the Insects will be sent to the Zoological Gardens, London, and others.

Lhotzky's observations gathered in vigour as the journey proceeded. Thus:

> Mr. ——, the gentleman who, with one or two more, claimed till the present time an exclusive monopoly and dictatorship over all information respecting the Natural History of New South Wales, and the transmission of specimens to the first-rate Museums in Great Britain—

Hinc illæ lacrimæ. Hence so much crying and sanguinary injustice I have endured in this Colony.

Later on:

Like Cain in the time of old stained with fratricide, I was marked with the guilt of two capital offences, which very few in the Colony (spoilt as it is by two bad and corrupted administrations) will ever forgive. I was a man without fortune and unconnected with Government. But enough of this jeremiad on Breadalbane Plains.

But the jeremiad had not ended. Referring to his stay at Limestone Cottage and his collection of minerals, he states:

I am rather at a miss to lay its results before the reader, because, my time and attention being taken up by selling wood and vegetables, my mind is consequently not quite in that equable state which a work of this kind requires . . . The writer of these lines was not deemed worthy to receive any official assistance whatever. My offence against the Government was indeed an inexpiable one. I reminded Governor Bourke and Mr. McLeay nearly two years since of a salary which was and continues to be voted for a dead man and I petitioned that the vacant situation might be bestowed on me *pro tempore*.

On page 66 we read:

I wandered happy upon the adjacent hills and gulleys, with the confident anticipation that so extensive a collection as I was able to make would gain, after my return, the approbation of the two persons who command our Colonial Museum; an expectation which, however, was altogether frustrated by reason of the old (but in this instance greatly misapplied) principle: *Amor incipit ab ego.*

Lhotzky suggested that, receiving no answer from the Colonial Museum to his letters offering his collections, he would send them to England to have them investigated. However, Mr. Iredale remarked that the last record he had concerning Lhotzky was an offer by the authorities of the Berlin Museum to sell by public auction the duplicates of his collections made on that journey and at Sydney. The catalogue was dated December, 1836, and the sale was to have been in the following April.

Lhotzky's records were of the greatest value. He was a keen and accurate observer. Besides minerals and plants, his collections on the journey comprised about 3,000 specimens of insects and over 100 birds and quadrupeds. Snails and other fresh-water shells also engaged his attention and description. On the expedition snakes were found, and he states that for snake-bite the Manero aborigines scarified the wound and several persons sucked it.

After which they wrench [*sic*] out their mouths. This operation is repeated till the patient is exhausted.

January 17 was a very hot day and Lhotzky observes that on such a day snakes are to be doubly apprehended. As the men were standing at noon quite close to the tent, in an entirely open place, one such venomous monster, about seven feet long, suddenly appeared amongst them. It was one of those termed black snakes, but Lhotzky pertinently remarks that such name embraced many distinct species. This particular individual was reddish under the body, evidently a true black snake. Two days later, again in intense heat, another black snake was killed near the tent. This was of a different species, with a yellow belly, but the details are not sufficient to permit of identification. Amongst the birds described during the journey were the laughing jackass, mutton bird (apparently the chough), the wild turkey, the New Holland vulture of Dr. Latham, ducks, the magpie, the white cockatoo, hawks, the grebe (or diver) *et cetera.*

One morning Lhotzky heard at daybreak "the singing of a little bird, the tones and strains of which were very delicate and tender". He states that this is rather rare in Australia, the feathered tribe here not being very melodious. At the same time he had nothing but admiration for "the gay flute-like tones" of the magpie. An account is also given of the "platipus" on the Limestone River. One was wounded, but "the spurs of the monster" prevented too close an approach. Lhotzky wisely states that at that time of our colonization it would have been "a loss of time and quite out of place in New South Wales to make new Genera and Species". This task should have been left to the authorities at the British Museum and in Europe.

Unfortunately the journal was not completed. There was no doubt, however, that Lhotzky was one of the first discoverers of gold in Australia. From the Australian Alps he brought some auriferous sand. From the records of the Australian Museum we learn that a Mr. John Benson Martin was one who attended at Dick's (the silversmith's) to witness its reduction and the first button of Australian gold turned out of its crucible. Seemingly, Lhotzky's finding was prior to that of his compatriot, Strzelecki. Further, he claimed to have discovered and named the Snowy River. He closely followed Strzelecki in exploring Mount Kosciusko. In *The Sydney Gazette* of April 15, 1834, he wrote from Jirabombra, on Limestone Plains:

Visiting many of the stations scattered about the interesting and important downs of Menero [now known as Monaro], I crossed the Snowy River and brought my cart as far as Mutong, situated about 37° S. Lat. and 148° E. Long., and entered by Westall's opening the very heart of the Australian Alps.

He mentions ascending Mount William, from 6,000 to 7,000 feet, "by far the highest point ever reached by any traveller on the Australian Continent". From this point he discovered a very extensive plain, larger than those of Menero, and called by the natives "Omeo". He was informed by the only man of the Menero tribe who had been there, that it contained a lake larger than Lake George. Lhotzky referred to the Snowy River as 200 yards in width, and considered that, in view of its size where he saw it, it must soon become navigable.

Canberra.

Lhotzky's itinerary took him to Limestone Plains, now known as the Federal capital, Canberra. He stayed at Limestone Cottage, now Duntroon, in February, 1834. According to Lhotzky, the aboriginal name of the place was Kembery. The initial "K" in the spelling is more definitely indicative of its pronunciation than the ambiguous

English "C". Lhotzky applied the name "Kembery Plain" to the whole valley north of the Molonglo River between the Ainslie Range and Black Mountain. With curiously prophetic vision Lhotzky said that at Limestone:

> At no distant period a fine town will exist, uniting Spencer's Gulph (by means of the Murrumbidgee), Sydney and Twofold Bay.

Lhotzky's journal gives a most interesting account of Canberra in the early days of habitation. Altogether he stayed six days at the Limestone Cottage. We read of the Quinbien Creek (now called Queanbeyan River) and other well-known places, the spelling of the names differing very materially from that now in use. Lhotzky's visit to Limestone Plains is of particular interest to myself, as my grandfather was the first resident land-owner about Canberra or in the Federal Capital Territory. Lhotzky visited my grandfather's place ("Springbank") and made some observations upon it in an excursion towardsd Ginninderra. He describes the locality as "a Tempe-like spot". About the middle of the plain there was

> a conspicouus conical mass of rock—McPherson's Sugarloaf, composed of serpentine of a fine greenish colour, which when polished would at some future period adorn the edifices of Limestone.

I never identified this Sugarloaf; the name has certainly long ago passed into oblivion. Black Mountain, however, was part of my grandfather's holding, and the quarries on the slopes of that mountain have yielded much stone used in building in the vicinity. So once again Lhotzky displayed prophetic vision. His narrative of the visit to my grandfather's place gives remarkable temperature observations during the day, culminating towards midnight in a dreadful storm of wind and rain

> which frightened the larger spiders and triantelopes, which were among the beams of the roof, and drove them into the room. I was lying in my bed and just observing through the window the fine effect of the lightning upon the plain, when one of the above-named gentry, nearly as large as a child's fist, fell *sans cérémonie* upon my face. I could not forbear whistling between my teeth "Oh the joys of travellers".

Mr. Keith C. McKeown, in his delightful book on "Spider Wonders", tried to trace the origin of the term "triantelope". His first record was its use by a British entomologist in South Australia in 1872, but we see from this narrative that it was employed many years prior to that date. The Arachnids so designated are species of Huntsman spiders of the genus *Isopeda*.

The Aborigines.

Lhotzky stated that some natives whom he encountered at Breadalbane Plains during this journey and who belonged to the Pajong tribe, informed him that they hunted upon the Yass plains but did not go as far afield as the Limestone Plains. Of the natives about Canberra (Kembery) Lhotzky says simply that "They are now no more". This was not quite true, as many aborigines continued to frequent Canberra and were not extinct for another thirty years. The reason why Lhotzky saw none might easily have been that in the course of their nomadic wanderings they were elsewhere at the time, or they were in concealment. It is, however, quite possible that the local tribe had somewhat disintegrated. In the Monaro district he encountered the "Menero" tribe, who were wont to visit Kuma (Cooma) station—sometimes sixty or seventy in number. He states:

> It is already very weak, consisting of about fifty men. They are entirely tame (indeed not civilised but currupted) and wander as far as Yass and Limestone Plains.

F. W. Robinson states that:

> Other tribes, less affected by the white man, which lay to west and south, were the Kunora or Gundanora and the Omeo, who were numerous, but very shy.

From the Menero blacks Lhotzky obtained a vocabulary of 160 words. These were supplied by young natives between the ages of thirteen and nineteen. The older blacks were much more reticent. In 1839 Lhotzky published, in *The Journal of the Royal Geograpical Society of London*, "Remarks on a Short Vocabulary of the Natives of Van Diemen's Land and Menero Downs". He also published "The Song of the Women of the Menero Tribe". This was an actual song, published in usual musical style.

There can be no question as to the interest and value of Lhotzky's observations on this trip, but one curious mistake occurs. Describing what were obviously grebes, he stated that there was no native name given to them (which was manifestly an error), but he considered that they belonged to the family of Pygopodes—which happen to be snake-like, legless lizards. Probably he intended Podicipidæ.

Lhotzky now engaged in political activities, and in 1834 signed a requisition to call a meeting in connexion with the appropriation of the land revenue; but he does not seem to have continued with such affairs. In the following year, however, he published a pamphlet, "Illustrations of the Present State and Future Prospects of the Colony of New South Wales", over the *nom-de-plume* of "Impartial Observer". On March 25 *The Sydney Gazette*, in a subleader, reviewed the pamphlet and referred to it as remarkable, having been written by one who "up to a little more than two years ago knew scarcely a word of the English language . . . We would rather peruse his book than abuse his politics". However, it takes the writer to task for a sneer "quite out of place" at Governor Darling's expense, and an undeserved attack upon Major Mitchell in connexion with his recent expedition into the interior.

In 1835, in Number II of *The New South Wales Literary, Political and Commercial Advertiser*, which was published by Lhotzky, we find a note entitled "Australian Geography. Private Interior Discovery". This dealt with a trip by Mr. C. Coxen, who was brother-in-law to the famous ornithologist,

John Gould. Coxen had come on behalf of the London Zoological Garden, and in December, 1834, had started from the Hunter River and travelled along the banks of the Namoi River, reaching 100 miles beyond Sir John Jamison's station. He brought back an excellent collection of birds, although the number stated as new to science was very greatly exaggerated—if, indeed, any at all were undescribed. Lhotzky apparently wrote the note with undue optimism.

Lhotzky's exploring fervour revived again in this year, and on October 23 he wrote to the Secretary of State, expressing his willingness to undertake another expedition into the interior and requesting that an order be sent to the Governor of New South Wales to provide him with the necessary number of assigned convicts. He stated also that he would be very thankful for any other facilities of carts, bullocks or horses he might receive out of the Government stores or herds. Here the title M.D. appears with Lhotzky's signature. On April 16, 1836, Baron Bulow wrote to Lord Glenelg, stating that Dr. Lhotzky, a German naturalist, residing at Sydney, was about to undertake another journey into the interior of the colony, where he wished to make collections for the Royal Museum at Berlin, and had transmitted to him a letter addressed to the Secretary of State for the Colonies; with the request that it be forwarded and support be given to the petition therein contained. Bulow, who was the Prussian Minister, recommended them to His Lordship, if he considered them admissible, being unable himself to judge whether circumstances permitted compliance with the wishes expressed. Lord Glenelg (April 22, 1836) wrote then to the Governor, Sir Richard Bourke, enclosing copies of Bulow's and Lhotzky's letters. Glenelg stated that of course it would be impossible to comply with the application, but that he had informed Baron Bulow that the Governor had been requested to afford Lhotzky any facilities which it might be in his power to accord consistently with established regulations. A further letter from Lord Glenelg to Sir Richard Bourke (November 10, 1836) requested the Governor to inform Dr. Lhotzky that he had received his letter of May 2, in which he sought remuneration for his discoveries in the interior (namely, his journey to the Southern Alps), but that as he had not forwarded his application through the Governor, he (Glenelg) had to defer his decision until the Governor furnished a report. In a note on the original of this dispatch it was stated that it was impossible to submit a report, as Lhotzky had removed to Tasmania.

Lhotzky in Tasmania.

Lhotzky now removed to Tasmania, and the turmoil attendant upon his New South Wales residence was mere pleasantry in comparison with his Tasmanian sojourn. A Tasmanian paper castigated him unmercifully. At the same time he was *persona grata* to Sir John and Lady Franklin when the former was Governor of Tasmania. Lhotzky had letters from them both. One from Sir John apologized to Lhotzky for not being able to offer him the full hospitality of Government House, as, owing to unforeseen circumstances, all spare accommodation was occupied, but, if Lhotzky cared to pitch his encampment in Government House grounds, he was most welcome to do so. The Governor assured him of a cordial welcome to his table at all times. But *The Sydney Gazette*, his erstwhile keen supporter, denounced him as "prince of humbugs" and stated that he had to run away from Sydney. *The Colonial Times* for October 18, 1836, gives in the shipping news intimation of the arrival of the barque *Francis Freeling* from Sydney with the following passengers: Mr. Hesse, Colonel Snodgrass, Mr. Arnold and Dr. Lhotzky. *The Tasmanian and Australasiatic Review* of October 21 states that:

> This very talented naturalist and enterprising traveller [Dr. Lhotzky] arrived here by the "Freeling". We perceive, in his advertisement in our third page, that a beautiful drawing of the barrier tiers, between Eastern and Western Australia, called by the Doctor "The Australian Alps", is very shortly to be raffled for and we have no doubt the people of Tasmania will evince the same liberality as was afforded by their Australian brethren towards a stranger of talent and enterprise.

It was mentioned that this view of the Australian Alps, near the pass leading from the Port Phillip country towards Sydney, was taken during the doctor's journey. It was remarked that four similar drawings were raffled for in Sydney and the shares of the first were taken in the short space of five days.

The Sydney Gazette, on November 1, 1836, continues its tirade:

> This celebrated literary character, has, we are happy to announce to our readers, arrived at Hobart Town. As the inquiries after the Australian traveller have been numerous, we are glad to appease the excited feeling, which his premature and unexplained absence has given rise to. We should, however, have been particularly happy had the doctor called at our Office, previous to his going, as we could have given him some information much to his advantage. As it is we must put up with the affront the best way we can.

There can be only one explanation for this outburst, and that is that the impecunious doctor left his financial obligations unabsolved. Now we find the doctor publishing a weekly tract called "Information for the People". The first issue contained sketches of Tasman's Peninsula. Other issues dealt with the Tasmanian Literary Institution, the Orphan School and the Public Garden of Hobart Town. It was also intimated that Dr. Lhotzky's collection, made in Tasmania, would be ready for public inspection. In one of the issues the doctor mentions that there was one object connected with the memory of Colonel Snodgrass, late Acting Governor of Van Diemen's Land, and that was the stone bridge in Argyle Street. It had been projected, planned, designed, estimated and ordered to be erected for many years past. But when they (including Colonel Snodgrass) arrived in Tasmania, there were only a few men amusing themselves, as

it were, cutting a couple of stones for the arch. However, the man of St. Sebastian did not like humbug. In a very short time twenty men were employed, then forty, and so on. When Snodgrass surrendered the government, the fine arch had grown up, like by magic, and only the keystones were required. Lhotzky said that the building should be called and installed as "Snodgrass's Bridge", but "it is ours to suggest and the people's to adopt".

In January, 1837, *The Sydney Gazette* returns to the attack with studied venom. It states that:

> Lhotzky has got hold of the editors of the different journals—his invariable plan—who give him an occasional puff. We wish we could join with them hand in hand in doing the same thing. This, however, we cannot do conscientiously. We don't like the man's principles. He left our office without paying us a farewell visit, much to our annoyance, for his name, unfortunately for the proprietors, stands conspicuously upon the ledgers. We also hear of the very disinterested way in which he bestowed his patronage and distinction, by borrowing from his friends a trifle of thirty shillings or so, which we suppose slipped his memory before he left for Hobart Town, or else he might have settled these little sums. Perhaps on his return he intends to do so. We hope he may. If he does not, we must put up with the loss and make up our minds as to whom to give credit in the future—that's all.

Lhotzky, however, continued on his way. We find him, in 1837, advocating the erection of a monument to Tasman in Tasmania. The Orphan School at New Town in Tasmania is trenchantly criticized by him, in regard both to the clothing of the inmates and to the blankets, in which he found needless extravagance. He found also that the tables were scrubbed too often. He likewise made reference to the many negroes who had been transported to Van Diemen's Land suffering bitterly from the cold through want of adequate clothing. This was especially noticed by him when negroes were assigned to him and he had to clothe them at an expense that was unwelcome to him. In February, 1838, we find that the doctor's museum was transferred to the Mechanics' Institute, with consent of his subscribers, "which bids fair for his speedy return to his native land". On December 1, 1838, in the *Proceedings of the Royal Asiatic Society*, it is stated that there was read a notice by Dr. Lhotzky of a New Zealand grammar and vocabulary compiled by the Reverend T. Kendall from the manuscript of a deceased gentleman. Mr. Kendall had begun printing the work in Sydney, but he having also died, it came into the possession of Dr. Lhotzky, who purposed publishing it in England. It was noted that the grammar fully elucidated the philosophy of the language and the vocabulary evinced its copiousness, especially on natural history subjects. It was remarked that the list of adverbs was extensive and comprised many of a complex kind, not existing in modern languages. The grammar also contained specimens of the native songs, some of which had already appeared in that journal. It was stated that the New Zealanders were of Malay origin.

In January, 1839, in *The Asiatic Journal and Monthly Register* it was stated that Dr. Lhotzky, who had resided for six years in the Australian colonies, was about to deliver three lectures on Australia before the Marylebone Literary Institution in England. It was added that the interest now excited by these colonies could not fail to make the lectures attractive. In the *Proceedings of the Royal Asiatic Society* for March, 1839, we note that the first paper read was one by Lhotzky concerning a recent discovery of ruins on Ascensis, one of the Caroline Islands. It was mentioned that some account of those remains had been published by Lhotzky in Sydney in 1834, particulars having been gathered from a man who had spent some months on Ascensis. It would appear that Lhotzky arrived in England in the year 1838. In *The True Colonist* of May 17, 1839, after his departure from Tasmania, a mild eulogy was printed concerning Lhotzky's paper dealing with the New Zealand grammar. This was a Tasmanian paper, and the mild praise was followed in the issue of May 24 by the most violent vituperation. He was stigmatized as an impostor and the author of slanderous misrepresentations in the English Press against the Australian colonies. The writer attempts to neutralize his efforts to injure the reputation of the people who treated him with so much kindness and, after they had become disgusted with his impudent tricks and impostures, furnished him with the means to leave the colony, where he could no longer make a living by his swindling. The article states that Lhotzky had been exhibiting his stolen wares before the Asiatic Society in London, and the report of the proceedings prompted a letter from a gentleman whose generosity was often taxed to supply Lhotzky's wants, and who, like everyone who did a kind act to this "character", received in return only insult and abuse when he would no longer comply with the impudent exactions and importunities of the doctor. The writer was astounded at the Asiatic Society being imposed upon, as Lhotzky knew as much about the language of the New Zealanders as he did about that of the Brobdingnags. The work was purloined from the Kendall family, but doubtless the "modern Dousterswivel" would get a good price for the manuscript and turn his plunder to some account. The writer stated that he had seen the manuscript which the doctor purloined, at Port Arthur, where he performed, or tried to perform, similar exploits, in the shape of frauds on the Government. In most of these he was frustrated by the lynx-eyed vigilance of Captain Booth. For some of them Lhotzky narrowly escaped the justly merited fate of obtaining from Her Majesty's judges letters of denization to render him fully entitled to all the privileges of those for whose accommodation Her Majesty's penal settlement at Port Arthur was established. Had Lhotzky met his deserts, instead of being in a condition to extort money from persons who returned to England with the fruits of their industry (earned in these colonies) by threats of holding them up in the English papers as returned convicts he would have himself been enduring well-

merited punishment as a convict at Port Arthur. The writer of the diatribe publishes a letter from one "J.S.", who contemplated proceeding to England, where it was hoped that he personally and publicly would expose in his true colours the "self-styled doctor" to those upon whom he was practising his impositions. "J.S." stated that the Reverend Mr. Kendall had lent the manuscript of the New Zealand grammar to Lhotzky, who had borrowed it under a pledge to return it in a few days. To this manuscript was appended a long list of names of gentlemen who proposed being subscribers to its publication, the amount of their subscriptions being about £200. Lhotzky refused to return the manuscript, even when offered a share in the profits of its publication, and also when threatened with an action for its detention. He was impervious to representations as to the necessity for its restoration to the family. Lhotzky admitted that it had been lent to him, but said that it had been lost and that in any case it was useless for any purpose. "J.S." added that on Lhotzky's arrival in Tasmania he showed the writer a manuscript history of the colony by Governor Collins, which, he said, he had "purchased from a prisoner" when staying at Port Arthur—another example of his perfidy.

In spite of all these vicissitudes Lhotzky did some very good scientific work. In botany the myrtaceous genus *Lhotzkya* was established by J. K. Schauer in 1835. It was named after Lhotzky, and specimens were introduced into England in 1843. Additional species were recorded in 1839, 1844 and subsequently. Some are dwarf shrubs with pale yellow or violet flowers; one is termed the Snow Myrtle of Victoria. There are at least ten species from the Swan River and elsewhere in Western Australia, South Australia and Victoria.

Here we may take our leave of the turbulent doctor. He was a man of many attainments, and an indefatigable naturalist; but his perpetual lack of financial resources made him unscrupulous in his monetary dealings. Everywhere admiration for his accomplishments eventually was replaced by anger and hostility on account of his financial dishonesty.

Reports of Cases.

THREE CASES OF RENAL FAILURE.

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It is only to be expected that when severe damage is inflicted on an intricate organ like the kidney, charged with the vital task of keeping constant the composition of the internal environment, there will be produced a kaleidoscopic group of symptoms which will vary considerably from case to case. These three cases are instructive in that they illustrate some of these variations. Furthermore, one of them suggests the possibility of an unusual form of excretory disturbance, namely, renal failure of nervous origin.

Case I.

A labourer, aged 26 years, was admitted to the Royal Prince Alfred Hospital on May 5, 1937, complaining of general weakness, dizziness, shortness of breath, swelling of the feet and frequent headaches, for a period of four weeks. For six months he had noticed vague signs of ill-health—rapid fatigue, some loss of appetite, occasional headaches and mental depression. However, he continued doing heavy manual work until three weeks before his admission to hospital, when he was compelled to stop by the symptoms mentioned above. Weakness and lassitude had been growing worse, while dyspnœa and dizziness, which appeared only on exertion, had also been increasing; at the time of his admission they were noticed even when he was sitting up in bed. His feet had been swelling slightly, and chiefly at night. There was no œdema of face or hands. Headaches had been severe, frontal, and worse in the mornings. There was no impairment of eyesight. No fits or twitchings occurred. The appetite was poor, there was no vomiting, and the bowels were regular. No other abdominal, nervous, or cardio-vascular symptoms were present. There was some frequency of micturition; five or six times during the day and two or three times at night. This he had noticed all his life. No scalding and no hæmaturia was present. His previous health had been good. He had had no illness suggestive of streptococcal infection or nephritis. Nothing was learned from the family history with regard to renal or cardio-vascular disease.

The great pallor of his mucous membranes was striking. Pronounced arterial pulsation was visible in the neck. His face was not puffy, but there was moderate œdema of the feet, ankles and sacrum. The pulse was rapid and regular in time and amplitude. The vessel wall was not palpable. The maximum blood pressure was 195 and the minimum 100 millimetres of mercury. The apex beat was in the fifth left intercostal space, 1·25 centimetres (half an inch) outside the midclavicular line. Auscultation revealed the presence of gallop rhythm, a faint blowing systolic murmur at the apex, and an accentuated second sound at the base. Ophthalmological examination revealed blurring of the disk edges, œdema of the retina and small streaky hæmorrhages scattered over both fundi. The retinal arteries were very slightly thickened. The tongue was furred but moist, and the fauces were normal. No further abnormality was detected in the alimentary, respiratory or nervous system.

Examination of the urine revealed the following facts. The colour was pale, the specific gravity was 1,005, its reaction was acid. It contained a considerable quantity of albumin. No other abnormality was detected chemically. With the microscope a few hyaline and granular casts and a few red blood cells were seen. The specific gravity and other findings remained practically the same throughout the illness.

A blood count made at the time of his admission to hospital revealed 1,910,000 erythrocytes per cubic millimetre; the hæmoglobin value was 37% and the colour index was 0·9. The blood urea concentration was 316 milligrammes; the creatinine content was 13 milligrammes, the calcium content was 8·9 milligrammes, the chloride content, in the form of sodium chloride, was 527 milligrammes, and the carbon dioxide combining power was 47 volumes, all per 100 cubic centimetres of blood.

The day after his admission to hospital the patient began to vomit, and continued to do so two or three times per day until his death. This symptom was most persistent and did not yield to any of the usual measures. Nevertheless, for the first three weeks he was able to maintain an adequate fluid intake. During this time he felt remarkably well and complained of no symptoms other than vomiting and headache, which yielded to aspirin and hypertonic saline solutions given *per rectum*. The œdema gradually subsided, only a small area being left over the sacrum. In spite of full doses of iron, the blood count continued to fall. The blood urea and creatinine maintained a steady

upward trend. On May 27 he became mentally confused and thereafter sank through drowsiness into stupor. The fluid intake and output consequently fell, the pulse rate rose steadily, and breathing became deep and hissing. On May 31 the blood urea was 630 milligrammes, creatinine 23 milligrammes, and the carbon dioxide combining power 25 volumes, all per 100 cubic centimetres of blood. The chloride content of the blood had fallen to 511 milligrammes per 100 cubic centimetres. The patient died on June 2, no twitchings or convulsions appearing at any stage.

At autopsy, the left kidney was found to be represented by a series of thin-walled cysts. Chronic nephritic changes were seen in the right kidney to a great degree, with thick, adherent capsule, gross granularity, and narrowed cortex containing a few cysts. The heart was moderately hypertrophied with pallor of the left ventricle and "thrush-breast" appearance from within. The coronary arteries were normal. No abnormality was found in the aorta or other great vessels. There were slight bronchopneumonic changes in both lungs.

A diagnosis was made of renal failure caused by chronic nephritis occurring in a person endowed by nature with only one functioning kidney.

Case II.

A man, aged 35 years, a fitter and turner by trade, presented himself for examination on May 19, 1937. He complained of increasing weakness, breathlessness, and palpitation on exertion, over a period of two years. Severe occipital headaches, throbbing in the head, and attacks of dizziness had been noticed for the same time. His feet had swollen occasionally, usually in the evenings. Nocturnal frequency of micturition had been present for two years (day four to five times, night two to four times). For six months he had been troubled by attacks of blurred vision, occurring every few weeks and lasting several days. Between attacks, vision was normal. He had remained fairly active, however, until three weeks previously, when his symptoms became worse and he was forced to stay in bed. Breathlessness was very troublesome, and sometimes occurred even during rest. His appetite vanished, and he was afflicted with persistent nausea, frequent dry retching, and occasional vomiting. He also began to cough, and to bring up small amounts of white, frothy sputum, sometimes blood-streaked. Headache was severe and persistent. No fits or twitchings occurred, and there were no other symptoms. His previous health had been good. No similar attacks had occurred before, and he had had no illness suggesting streptococcal infection or nephritis. The only relevant fact in the family history was that his father had died of "blood pressure".

On examination, the patient looked like a man of fifty (though actually only thirty-five years of age); the hair was grey and the face was lined and sunken. The lips and ears were slightly cyanosed. Marked pulsation was visible along the line of the carotid vessels. Slight œdema of ankles and sacrum was present. The pulse was rapid and regular in time and amplitude. The radial arteries were grossly thickened and tortuous. The systolic blood pressure was 205, and the diastolic 170 millimetres of mercury. The apex beat was in the fifth left intercostal space, in the anterior axillary line, that is, about 12·5 centimetres (five inches) from the middle line. Auscultation revealed nothing abnormal except an accentuated second sound at the base. No murmurs nor irregularities of rhythm were present. The optic fundi displayed very narrowed, glistening retinal arteries, and nipped veins; no abnormality of the disks, and no hæmorrhages or exudates were seen. Apart from an enlarged, tender liver, no abnormality was detected in the other systems.

The urine was examined and found to be pale, the specific gravity being 1,012. Its reaction was acid, and it contained a large quantity of albumin. No blood, sugar or acetone was detected chemically. Microscopically, some hyaline and granular casts and a few red blood cells were seen.

On the patient's admission to hospital, a blood count gave the following information: the erythrocytes numbered 5,050,000 per cubic millimetre, the hæmoglobin value was 102%, the colour index was 1·0, the leucocytes numbered 17,400 per cubic millimetre, 81% being neutrophile cells. The blood urea was 157, and creatinine 3·3 milligrammes per 100 cubic centimetres of blood. X ray examination of the chest revealed pronounced uniform enlargement of the heart, and a shadow in the right upper lobe (possibly an early tuberculous lesion). Examination of the sputum revealed no tubercle bacilli.

The patient progressed rapidly downhill. Headache was frquent and severe, as were nausea and vomiting. These symptoms were somewhat relieved by rectal injection of a 50% solution of magnesium sulphate. The pulse was persistently rapid, and the blood pressure remained fairly constant until just before death, when it fell considerably. The temperature was remarkably low; it varied between 30·5° C. (95·0° F.) and 36·7° C. (98·0° F.), and was never over 36·92° C. (98·4° F.). Peripheral cyanosis was very striking towards the end, and the signs of congestive cardiac failure were manifest. Œdema increased steadily in the lower limbs and back until the patient was virtually waterlogged; diuretics and purgatives were without effect. The liver became larger and more tender, and numerous râles appeared at the lung bases. On June 15, the blood urea had risen to 333 milligrammes, and the creatinine was four milligrammes per 100 cubic centimetres of blood. Drowsiness appeared, and fluid output began to fall. The patient lapsed into stupor, and died on June 21. At no time did he suffer from twitchings or convulsions.

At autopsy the arteries, small, medium, and large, were seen to be grossly sclerotic, with many atheromatous patches. The right auricle was dilated, and the right ventricle was dilated and hypertrophied. The left ventricle was very much hypertrophied. The coronary vessels were grossly atheromatous. All the viscera displayed evidence of venous congestion, especially the liver. The kidneys were slightly smaller than normal, with some adherence of the capsule to their finely-granular surfaces, which were dotted with small hæmorrhages. On section, the cortex was somewhat narrow and the rental arteries were sclerotic. In the upper lobe of the right lung was a small patch of pneumonic consolidation. Small scars of old apical tuberculosis were present in both lungs.

A diagnosis was made of hypertension of the malignant type, with pronounced arteriosclerosis. A small patch of pneumonia was present, and renal and cardiac failure had occurred.

Case III.

A single woman, aged 22 years, was admitted to the Royal Prince Alfred Hospital on May 14, 1937, complaining of feeling generally "off-colour" for five days. The appetite was poor, and nausea and vomiting were troublesome. The bowels were regular, and no diarrhœa or abdominal pain was present. For three days only small amounts of urine had been passed and slight scalding had been noticed. No hæmaturia, no pain in the back, and no œdema was present. Some headache had been present since the onset. There was no dimness of vision. No fits or muscular twitchings occurred. There were no other symptoms. Her previous health had been good. There was no history of streptococcal infections nor of nephritis. No relevant family history was forthcoming, except that she was the mother of a healthy infant aged six months. Further questioning revealed that this illegitimate child had caused her much mental stress and physical hardship. Her parents had disowned her, and since the birth of the child she had been obliged to do hard manual work, and had lived under conditions of appalling squalor, most of the time half-starved.

On examination, the patient was seen to be ill-nourished, and looked exhausted and apathetic. No cyanosis nor pallor, and no puffiness of the face was present. No abnormal pulsations, and no œdema of the extremities or the back were present. The pulse was slow, and regular in time and amplitude. The vessel wall was not palpable. The systolic blood pressure was 145 and the diastolic 95 millimetres of mercury. There was no enlargement of the heart, and the heart sounds were normal. The tongue was furred but moist, and the breath was rather offensive. There was a small ulcer on the inner surface of the right

cheek opposite the third molar. Apart from a retroverted uterus, no other abnormality of any system was detected.

The patient passed no urine on the day of admission to hospital. The bladder was not palpable, and catheterization showed it to be empty. The blood urea was estimated and found to be 404 milligrammes, and the blood creatinine was 6·6 milligrammes per 100 cubic centimetres of blood. Four pints of normal saline solution with 10% glucose were given intravenously by the drip method, and on May 15 she passed a few ounces of urine. Examination of the urine revealed that the specific gravity was 1,015 and the reaction acid. A faint trace of albumin was found. No other abnormality was detected except some glucose (due to the intravenous injections of sugar). No casts or red blood cells were seen microscopically.

X ray examination of the renal tract revealed no stones, and after cystoscopy both ureters were catheterized easily. However, only a few drops of urine flowed. On May 16, the patient was feeling better and vomited less. Two pints of normal saline solution were given intramuscularly. Next day she was able to retain fluids, and was urged to drink as much as possible. From then on she improved rapidly; no further vomiting occurred, and she drank six to eight pints of water each day. Headache and malaise disappeared, and appetite returned as the urinary output rose. On May 19, the blood urea was 330 milligrammes, the creatinine five milligrammes, the calcium nine milligrammes, the chloride 409 milligrammes, and the carbon dioxide combining power 47 volumes, all per 100 cubic centimetres of blood. By May 24, the blood urea had fallen to 107 milligrammes, and the creatinine to four milligrammes per 100 cubic centimetres, and on June 1, the blood urea was 44 milligrammes and the creatinine one milligramme per 100 cubic centimetres. During this time the urine exhibited no abnormality except a faint trace of albumin. The specific gravity varied between 1,012 and 1,020. A urea concentration test performed on June 3 showed that her kidneys were capable of concentrating to 1·95% at the second hour, and to 2·05% at the third hour. Intravenous pyelography performed on June 10 revealed that the renal tract was free from abnormality and that the kidneys were functioning normally. Her systolic blood pressure was at that time 130 and her diastolic 85 millimetres of mercury, and her urine was albumin-free. She was discharged on June 11, apparently in normal health.

A diagnosis of urinary suppression, possibly of psychogenic origin, aggravated by starvation, was made.

Discussion.

Here we have three cases of renal failure, all in young subjects, and all of somewhat similar severity, but nevertheless with considerable differences. The renal failure in Case I was caused by primary renal lesions; in Case II it was due to arterial disease and complicated by cardiac failure; while in Case III it occurred in a subject with apparently normal vascular and renal apparatus. The symptoms and signs also varied considerably in the three cases. An attempt will be made to analyse briefly these findings.

Of all the symptoms exhibited by these patients, nausea and vomiting were undoubtedly the most prominent and the most troublesome. In Case I the cause was probably central, vascular changes in the medulla, accompanying the cerebral œdema which was almost certainly present, producing stimulation of the vomiting centre; hence the failure of locally-acting measures such as gastric sedatives. In Case II, a local factor probably played some part, namely, passive congestion due to cardiac failure. Certainly locally-acting drugs, such as small doses of tincture of opium and belladonna, were beneficial in this case. In Cases I and II hypertonic saline solutions given rectally gave some relief temporarily, no doubt by diminishing cerebral œdema. In Case III the cause of vomiting is more obscure. Cerebral œdema may have played a part, but there was little evidence of this condition. It may be that the high non-protein nitrogen of the plasma was in itself enough stimulus to the vomiting centre. Another possibility is that the nausea and vomiting were primary rather than secondary phenomena, manifestations of a general neurotic state, which served to aggravate renal failure by causing dehydration and loss of salt with consequent rise in the blood urea.

Headache was experienced in each case, but varied in severity. It appeared to depend upon the degree of hypertension, being severe in the first two cases, and comparatively slight in Case III, in which the blood pressure was raised little if at all. Tension in the meninges and meningeal vessels caused by raised intracranial pressure would appear to be the mechanism of this symptom. Hypertonic saline solutions given *per rectum* afforded great relief.

The mental state was interesting, all three patients exhibiting general mental dulness and apathy towards their surroundings. In the two fatal cases, this progressed steadily through drowsiness into stupor. How much these changes in cerebration were due to chemical changes in the *milieu intérieur*, and how much to vascular alterations and œdema of the brain, is difficult to say; but the fact that in Case III (in which there was little evidence of cerebral vascular disturbance) the mental state improved *pari passu* with the fall in blood urea, indicates that the chemical changes are largely, if not wholly, responsible for this impairment of function. In the later stages of renal failure, acidosis is usually pronounced, on account of failure of sulphate and phosphate excretion and also of starvation ketosis, and the resultant rise in blood hydrogen ion concentration, combined with the toxic action of enol-containing ketones, would seem to be responsible for the stupor or coma into which these patients sink, also for the hyperpnœa so often seen. The first patient, whose carbon dioxide combining power shortly before death was 25 volumes per 100 cubic centimetres of blood, was deeply stuporose, and developed deep, hissing respiration.

The variations in œdema in these three cases are instructive. The first patient, on admission to hospital, had moderate œdema, which gradually diminished. The second at first was slightly œdematous, but this condition increased tremendously as he became worse; while the third patient had no œdema at all. Œdema in Case I was probably caused by gross anæmia. There were no signs of congestive cardiac failure, and protein loss in the urine would not have been great enough to be responsible. The decrease in œdema was almost certainly due to dehydration. Case II in which such gross œdema occurred, was complicated by the presence of severe congestive cardiac failure. Though protein loss may have played some part, the chief cause was undoubtedly cardiac failure. The third patient behaved, in effect, like a person after double nephrectomy; here one would not expect œdema in these circumstances, none of the factors necessary for its production being present: no cardiac failure, no loss of blood protein, and no capillary toxin (as in acute nephritis). All this serves to emphasize the fact that even complete renal failure *per se* cannot produce œdema.

Anæmia was present only in Case I, in which it was severe and persistent, and was responsible for many of the symptoms; apart from œdema, it caused the patient's dyspnœa and palpitations on exertion, his lassitude and probably his dizziness. As this patient had been existing with very little active renal tissue for some years, there must have been considerable retention of nitrogen *et cetera* throughout this period. It is likely that the failure of his hæmopoietic mechanism was caused by this prolonged immersion in an abnormal environment. The second patient whose renal failure was secondary to vascular abnormality and comparatively recent, had only slight anæmia before death. Excretory failure was of such short duration in Case III that no damage to blood-forming tissue resulted.

The urinary findings in these cases were not unusual, except those in Case III. In the first two cases the urine was of low, fixed specific gravity, showing that the remaining renal elements were functioning at the limit of their capabilities. Moderate albuminuria and the presence of red blood corpuscles and casts indicated glomerular damage in the active renal tissue. Case III, however, presented no urinary abnormality apart from a trace of albumin. This excluded the presence of acute inflammatory change in the kidneys; acute nephritis, focal or diffuse, would have caused the appearance of red blood corpuscles and casts.

The blood chemistry was interesting if only from the magnitude of some of the figures; for example, in Case I the blood creatinine concentration was 23 milligrammes per 100 cubic centimetres shortly before death. The blood calcium was very little below normal in Cases I and III. The absence of twitchings may be related to this. The blood chloride was only slightly diminished, while the carbon dioxide combining power was only slightly depressed, except in Case I, in which it fell to 25 volumes per 100 cubic centimetres not long before death.

But the most interesting point is that the prognosis cannot be gauged by the magnitude of these figures alone. One would be inclined to give a grave prognosis in a patient with a blood urea of 404 milligrammes and creatinine content of 6·6 milligrammes per 100 cubic centimetres. Yet the third patient had these figures and made a good recovery.

This brief attempt at analysis of symptoms and signs is obviously incomplete and largely tentative, but it is always interesting and instructive to try and correlate physical findings with their underlying pathology.

A few words remain to be said about the cause of renal failure in Case III. What was it that suddenly put out of action the whole of her apparently normal renal epithelium? For one cannot believe her condition to be nephritic, in the absence of all the usual signs; there was no œdema, no lumbar pain, and no red blood cells or casts were in the urine. She behaved, in fact, as if both kidneys had been removed. In view of her history of privation, mental stress and starvation, one feels justified in postulating urinary suppression of nervous origin. Hypothalamic centres are known to exist which control water-metabolism in some obscure way, partly through the pituitary, and partly through the autonomic system. The kidneys are richly supplied with autonomic nerves. Very little is known even of the normal action of these nerves. It is quite possible that this unfortunate girl's failure of excretion was produced by means of this intricate system, whether by its causing glomerular ischæmia, or by more direct action on renal epithelium, it would be idle to speculate. With increasing failure of excretion came persistent vomiting, causing further upset of the internal environment, and the establishment of a vicious circle, which would probably have proved fatal but for copious parenteral administration of fluids and glucose. This explanation, though speculative, at least accounts for the findings. The influence of the nervous system on involuntary processes is often under-estimated.

Summary.

1. Three cases of renal failure in young subjects are described.
2. An attempt is made to correlate the chief findings with their underlying pathology.
3. The possibility is suggested of renal failure occurring through the action of the central nervous system.

Reviews.

HAIR AND SCALP.

THE well-deserved popularity of Dr. Agnes Savill's first edition of "The Hair and the Scalp" is evidenced by the fact that a second edition has been published within two years.[1]

In the preface to the second edition she mentions that the book has been lengthened by twenty pages and that much has been rewritten.

In the section on the diameter of the hair the author draws attention to the fact that flaxen hair is finer than dark hair, and that the hair on the head of women is, on the average, slightly thicker than that of men. Mention of the work of Dr. U. Matsuura is also well deserved, as he has shown that the state of the hair has a direct bearing on the previous health of the individual, and he has compared this with the well-known fact that poor health in a sheep is reflected in its wool. Two methods of staining hair infected with fungus are described, one for permanent mounting, and a simple method for out-patient work. A classification of the various types of *atrichia congenita* is given, and the ætiology and prognosis are described.

The section on *alopecia areata* is very fully written, practically all the theories of its ætiology being mentioned. Dr. Savill states, however, that almost all the advocates of the different theories can produce cases which have tended to support their contentions. Many new methods of treatment are described, including intradermal injections of methylacetylcholine, the administration of histamine to the patch with the aid of a galvanic current, dosage of the sympathetic with *Grenz* rays, and an American device for producing hyperæmia of the scalp by suction.

A paragraph on *acne conglobata* has been added and the technique for X ray treatment has been briefly detailed. Recent therapeutic measures for hirsuties are fully dealt with, and Dr. Savill states her preference for the slower treatment by electrolysis over the faster treatment by diathermy.

On page 175 a picture illustration is given of *fausse teigne amiantacée* under the newer title of a streptococcal infection, and attention is drawn to the fact that Dr. Sabouraud himself is responsible for this change in nomenclature.

Many new electrical and physical methods credited with hair-stimulating properties are mentioned throughout the book.

Three hundred pages are contained in the book, which has not only maintained but improved on its previous high standard. Over three pages of formulæ are to be found at the end of the book, and numerous excellent illustrations are included.

[1] "The Hair and Scalp: A Clinical Study (with a Chapter on Hirsuties)", by A. Savill, M.A., M.D., M.R.C.P.I.; Second Edition; 1937. London: Edward Arnold and Company. Demy 8vo, pp. 317, with illustrations. Price: 12s. 6d. net.

OTO-RHINO-LARYNGOLOGY.

MANY attempts have been made to produce small yet easily read and practical text-books in the special branches of medicine and surgery. "Diseases of the Ear, Throat and Nose", by J. Douglas McLaggan, represents a very fair attempt in this direction.[1] Perhaps a more elaborate description might be provided of some of the commoner procedures, such as full details of the method of production of local anæsthesia for the performance of proof puncture of the antrum, and of the procedure of puncture itself. Surely it is a mistake, too, to omit a full description of guillotine tonsillectomy. There must remain many workers who will not agree with the proposed abandonment of this method in favour of universal dissection. Some of the commoner operations, such as submucous resection of the nasal septum, are described in even a little more detail than the student or general practitioner might require, yet with insufficient fullness for the actual performance of the operation; while the operation for removal of nasal polypi, which so many general practitioners will perform, might receive a much fuller description, together with illustrations. Similar criticism must apply to the description of the operation of *paracentesis tympani*.

The work comprises an excellent general foundation for improved editions which should follow. For the present it contains an abundance of information which should help the medical student during his general curriculum. It is hardly adequate as a reference work for the general practitioner who wishes to perform a certain amount of ear, nose and throat surgery himself. It is quite inadequate as a text for the preparation for any of the special examinations in this subject.

[1] "General Practice Series. Diseases of the Ear, Throat and Nose", by J. D. McLaggan, M.A., M.B., F.R.C.S.; 1937. London: H. K. Lewis and Company Limited. Demy 8vo, pp. 346, with 9 plates and 135 illustrations. Price: 15s. net.

The Medical Journal of Australia

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All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: Initials of author, surname of author, full title of article, name of journal, volume, full date (month, day and year), number of the first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

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THE OPHTHALMOLOGICAL SOCIETY OF AUSTRALIA (BRITISH MEDICAL ASSOCIATION).

For many years, as readers well know, members of the British Medical Association in Australia have laboured under many difficulties when they wished to form sections or special groups of members who were interested in and practised special branches of medicine. It is true that sections for the study of special branches of medical knowledge could be formed, but those comprising the section could not refuse membership to any member of the British Medical Association who wished to join it. This provision proved to be an insurmountable bar to the formation of any special section that would comprise the whole of Australia. When officials of the Home Association visited Australia in 1935 for the 103rd annual meeting of the Association, their attention was drawn to the disabilities experienced by the Australian Branches in the formation of special sections. The Parent Body in its wisdom agreed to set its legislative machinery going so that the wishes of the Australian Branches should be met. The machinery has performed its slow and stately revolutions, and after more than two years special sections can be formed in Australia in a way of which Australians approve.

The first special section has been formed and has been called The Ophthalmological Society of Australia (British Medical Association). At the Adelaide congress in August, 1937, it was decided by ophthalmologists who were present that this section should be formed. One representative from each State was elected to membership of a committee, and this committee drew up rules which were submitted to a specially convened meeting in Sydney on March 23, 1938. After discussion of the rules it was resolved, on the motion of Dr. Ringland Anderson, seconded by Dr. Bruce Hamilton:

> That the members of the British Medical Association in Australia specializing in ophthalmology and present in person or by proxy incorporate themselves in a body to be named The Ophthalmological Society of Australia (British Medical Association), and that the rules as tabled be adopted as the rules of the society, and that this meeting be deemed the inaugural meeting of the society.

Sir James Barrett is president and Dr. James Flynn, of Sydney, is vice-president. The honorary secretary is Dr. Darcy Williams, of Sydney, and the honorary treasurer, Dr. Leonard Mitchell, of Melbourne. The members of the council are Dr. E. J. McGuinness, of Brisbane, Dr. Ringland Anderson, of Melbourne, Dr. Bruce Hamilton, of Hobart, Dr. G. Barham Black, of Adelaide, and Dr. Claude Morlet, of Perth. The objects of the society are:

1. The cultivation and promotion of ophthalmology and related sciences by all or any of the following:
   - (*a*) The periodical meetings of members.
   - (*b*) The collection and dissemination by or amongst members of scientific knowledge and the publication of articles relating to ophthalmology and the scientific proceedings of meetings.
   - (*c*) The promotion of research in ophthalmology with or without grants of sums of money out of the funds of the society or out of funds donated for that purpose.

(*d*) The establishment and maintenance of ophthalmological libraries and ophthalmological museums of normal and pathological anatomy.

(*e*) The establishment of prizes for the encouragement of scientific ophthalmic work out of the funds of the society or out of funds donated for that purpose.

(*f*) The arrangement of visits by ophthalmologists from abroad for the purpose of giving lectures and demonstrations.

2. To confer, cooperate and affiliate with other bodies having altogether or in part similar objects within the Commonwealth of Australia or elsewhere.

3. To provide an authoritative body of opinion on matters concerning vision and to promote the visual health of the community and to prevent blindness.

4. To make representations through the Federal Council of the British Medical Association in Australia, to the legislature and public officials and others on matters affecting the visual health of the community and the interests of members.

There will be ordinary, honorary and associate members. Those eligible for ordinary membership are all members of the British Medical Association in Australia who specialize in ophthalmology and whose qualifications are approved by the council. It will be necessary for intending members to submit to the secretary a completed application form and to be proposed for membership by two members residing in the State in which they themselves reside. So far, 120 applications for ordinary membership have been received. The council has appointed a literature committee, a research and scientific committee, an orthoptic committee, a visual conservation committee, and an industrial compensation committee.

The foundation of this society is the result of no sudden impulse; ophthalmologists in Australia have long felt the need for some means of doing those things that they have set out as their objects. That the present enthusiasm will wane is therefore unlikely. It is but natural that men and women studying and practising the same specialty should by the gathering of themselves together increase their knowledge and improve their efficiency. At the same time we would lay special emphasis on the other ways in which this new body can serve the community. That service of this kind is intended is clear from the titles of some of the committees that have been formed. Finally, we welcome an intramural society with the hope that its success may induce some of the existing extramural bodies to become part and parcel of the British Medical Association.

Current Comment.

THE ELECTROCARDIOGRAM IN MITRAL STENOSIS.

In recent years there has been a tendency to minimize the effect of valvular lesions and to regard infection of the myocardium as almost the sole determining factor in the course of rheumatic heart disease. The older view was to regard the valve lesion as the more important handicap. Truth lies between the two extremes, and many patients with healed rheumatic carditis behave as if the valve lesion and not the myocardial disease finally caused failure and death. Kurt Berliner and Arthur Master have recently correlated the electrocardiographic and *post mortem* appearances found in sufferers from uncomplicated and complicated mitral stenosis.[1] They make the statement that electrocardiography demonstrates the specific effect of each valvular lesion on the heart hardly less well than fluoroscopy. The report is based on a series of 113 consecutive patients, concerning whom 265 electrocardiograms have been recorded. Patients in whom arterial hypertension was associated with mitral stenosis were carefully excluded, also patients presenting a large hydrothorax or any other factor capable of altering the axis of the heart. The patients were subdivided according to the associated condition of the tricuspid and aortic valve as determined by *post mortem* examination. Further, one-sided preponderance in the electrocardiogram was compared with the anatomical changes in the ventricles found at autopsy. Their results are interesting not only as regards the controlled evaluation of the textbook cardiographic signs of mitral stenosis, but also as to the degree to which these signs are modified by other valve lesions, and especially an associated tricuspid insufficiency. Notching of the *P* wave, which occurs more frequently in mitral stenosis than in any other condition, was found in 61 of 69 patients with mitral stenosis and regular sinus rhythm. The average height in Lead II of this *P* wave was 1·6 millimetres; in other words, a great increase in the amplitude of the *P* wave is not frequent in cases of uncomplicated mitral stenosis. When, however, hypertrophy of the right auricle existed in addition to that of the left, a high *P* wave of from 2·0 to 3·5 millimetres was found in those patients who had not suffered from fibrillation. The highest *P* waves.

[1] *Archives of Internal Medicine*, January, 1938.

3·5 millimetres or more, were observed in cases of mitral stenosis associated with tricuspid stenosis. The authors point out that it is well to remember at this point that a constant relation between the height of the *P* wave and the degree of auricular hypertrophy can never be expected any more than the height of the *Q R S* complex is a true index of the degree of ventricular hypertrophy. Indeed, it is surprising that the exceptions are not more frequent, as the degree of functional activity of the muscle must play a large part. The search for electrocardiographic signs of auricular preponderance failed completely. When a left auricle was found at autopsy to contain a rheumatic lesion, the *P* wave of the electrocardiogram was indistinguishable from that in which the auricular wall was normal. In the group of cases of mitral stenosis with aortic insufficiency the average height of the *P* wave was no greater than that seen in pure mitral stenosis. In contrast with the former group it seems that the disease of the tricuspid valve affects the relative size of the auricles to a considerably greater degree than disease of the aortic valve. Comparison of *P* waves in patients with mitral insufficiency and patients with mitral stenosis revealed the following differences: a widening of the *P* wave was rarely found with myocardial insufficiency and notching was observed in only half the cases. The advent of auricular fibrillation was related much more distinctly to tricuspid valve disease than to aortic valve disease in association with mitral stenosis. A statement is made that ventricular preponderance shows more clearly than any other electrocardiographic signs the profound effect which a valvular lesion exercises on the dynamics of the heart. Comparing the patients who had a tight, narrow mitral stenosis with those in whom the narrowing was moderate or slight, these authors showed that hypertrophy of the right ventricle was a rule in the former group, and that in such patients right ventricular preponderance was apparent. Ventricular preponderance depends, then, on the degree of stenosis, and this, for a long while expected, represents a definite increase in our knowledge. Outright left ventricular preponderance was never found in a case of mitral stenosis with or without mitral insufficiency unless it was complicated by aortic valvular disease. When this complication existed it tended to swing the balance of the ventricles towards the left side. The preponderance clearly depended upon the extent of the leak in the aortic valve. In aortic insufficiency with high pulse pressure there was left ventricular preponderance whether the associated mitral stenosis was slight or pronounced.

In a review of 93 patients with mitral stenosis, simple or complicated, it was found that forty-five, or about half, showed only right ventricular preponderance. Mitral insufficiency revealed itself as having less effect on ventricular preponderance than mitral stenosis. It was noted that mitral valvular disease *per se* had little influence on intraventricular conduction. It was noted that increase of voltage in *Q R S* was not a common finding in mitral stenosis. A high degree of right ventricular hypertrophy was frequently found without any increase in voltage, as is frequently the case in congenital heart disease.

Berliner and Master conclude that the left ventricle is the one which contributes most to the voltage of the ventricular complex. As was expected, inversion of the *T* waves in the third lead was particularly frequent, especially in the patients with tight "button-hole" stenosis. An interesting comparison was made between *post mortem* findings of the ventricles and the corresponding electrocardiograms. In 89% of 85 such patients there was agreement between electrocardiographic preponderance and anatomical preponderance.

Perhaps the most valuable lesson to be learned from this study is the realization of the importance of the associated valvular lesion. At the bedside the associated tricuspid lesion is difficult to recognize, although it is known that lesions of the mitral valve alone are rarer than combined lesions of mitral and tricuspid valve. In other words, a "typical" case of mitral stenosis is not a pure case of mitral stenosis, and the "typical" electrocardiogram of mitral stenosis is the electrocardiogram of a combined lesion. Notching of the *P* wave remains a most characteristic electrocardiographic sign of mitral stenosis. Right ventricular preponderance, however, was found in less than half the patients examined. On the other hand, left ventricular preponderance absolutely excludes the diagnosis of uncomplicated mitral stenosis, provided hypertension can be excluded. Thus a soft diastolic murmur at the base of the heart in a patient with clear-cut mitral stenosis, but in which left ventricular preponderance is revealed in the electrocardiogram, is not a Graham Steel murmur, but indicates some degree of aortic insufficiency. If, with the left ventricular preponderance, there is high voltage of the *Q R S* and the duration of this complex is 0·1 second with only moderate notching of the *P* wave, the diagnosis of aortic insufficiency *plus* disease of the mitral valve may be assumed. The electrocardiogram of mitral insufficiency is apparently non-characteristic. It is noteworthy that in the present series auricular fibrillation never occurred in association with mitral insufficiency, even if aortic insufficiency coexisted; and in such patients, too, the *P* wave of the tracing was different; notching was present in only half of this group and right ventricular preponderance was never seen.

The authors appear to have established the fact that the altered anatomical relationship of the ventricles in valvular disease far outweighs other factors, such as the type of chest and the height of the diaphragm, which are theoretically capable of altering the form of the ventricular complex.

Abstracts from Current Medical Literature.

MORBID ANATOMY.

Shock.

Virgil H. Moon (*Archives of Pathology*, November and December, 1937) writes on the mechanism and pathology of shock. Various hypotheses have been advanced to explain the condition, but all have failed to withstand subsequent investigation. Crile championed the theory of "vasomotor exhaustion", but it has been proved that there is actually an increase in vasomotor activity for several hours during shock, and that it declines only after the blood pressure has been reduced below 50 millimetres of mercury. In shock the arteries are not relaxed, but maximally contracted, and it may be concluded that the heart, arteries and the nerves controlling their action are not primarily involved. Henderson advanced the theory of acapnia, which was, however, disproved by showing that when the blood carbon dioxide was maintained at or above normal levels the susceptibility to shock was not diminished. Moreover, there is no hyperpnœa following severe wounds. Fat embolism, though resembling shock, differs from it in that the venous pressure is raised and it affords no adequate explanation for the mechanism of shock. The hypothesis of the decrease in the alkali reserve cannot be substantiated, as, although shock is accompanied by an acidosis, the evidence indicates that this is the result of deficient oxidation due to a reduction in circulation, that is, that it is a result and not a cause of circulatory deficiency. Experience during the war convinced many that the absorption of products from injured tissues was a major factor in the production of the circulatory deficiency which is the essential feature of shock. This deficiency frequently was out of all proportion to the apparent severity of the wound, and subsided in a remarkable fashion after amputation of the mangled limb or the *débridement* of other wounds. It has been shown that whereas after hæmorrhage there is a dilution of the blood, shock is characterized by hæmoconcentration due to stasis of blood in capillary areas and transudation of plasma. The hæmoconcentration is progressive and tends to be proportional to the degree of shock. In severe shock the vascular structures are incapable of retaining colloid solutions or even whole blood. This is due to the loss of tone of the capillaries and an increase in the permeability of their endothelium, and is brought about by the direct action of substances absorbed from injured tissues or other similar agents. Thus shock may be defined as a circulatory deficiency not cardiac and not vasomotor in origin, characterized by decreased total blood volume, decreased volume flow, and by hæmoconcentration.

Tissue Culture of Intracranial Tumours.

L. B. Cox and M. L. Cranage (*The Journal of Pathology and Bacteriology*, November, 1937) review briefly the literature on the tissue culture of intracranial tumours, and record their results of cultures studied in the Baker Institute, Melbourne. Not only have the authors employed the morphology of stained and unstained preparations in the distinction of true migrating tumour cells from fibroblasts and monocyte-macrophages, but they have used the capacity of the cells to segregate trypan blue. They observed migration of cells from four out of nine meningiomata. The cells of two of these were judged to be truly neoplastic; they resembled the tumour cells of the stained sections and did not segregate trypan blue. The migration was in one a sheet formation with mitotic figures, in the other polar cells. It could not be proven that tumour cells were present in the migration from the other two tumours. Migration of small polar cells was obtained from one out of four auditory nerve tumours. These cells were regarded as tumour cells, as they resembled those in section and did not segregate trypan blue. Of three hæmangioblastomata of the cerebellum cultured, migration was observed in two. Chains of polar cells emerged from one which segregated trypan blue but lightly, and were thought to be tumour cells. A dense sheet of growth, unlike that observed in any other tumour, was obtained from the one medulloblastoma cultured. It consisted of small undifferentiated cells, many of them being in mitosis. Migration was obtained from two out of seven astrocytomata. In one of these, a piloid astrocytoma, the migrating cells were true astrocytes of polar form and could be impregnated with gold sublimate. Migration occurred from two out of three glioblastomata. The migrating cells were polymorphic, as observed in the tumour sections. They did not segregate trypan blue, nor did they become impregnated with gold sublimate. The appearances of the cultures were characteristic. Of five pituitary adenomata cultured, a scanty migration of small unidentifiable cells occurred from two tumours of the chromophobe type. In one carcinoma metastasis an unsuccessful attempt at culture was made. The authors conclude that when a great number of intracranial tumours have been cultured and studied, the types of tumour cell will be better understood. Much that is unsatisfactory in the present classification may then be eliminated. The migrating cells from fibroblastomata and gliomata retain in a remarkable way the appearances of the tumour cell type. They may undergo mitosis, but show no evidence of differentiation.

Incidence of Metastasis of Malignant Tumours in the Adrenals.

D. A. Glomset (*American Journal of Cancer*, January, 1938) has analysed the results of 4,000 consecutive autopsies performed at the department of pathology of the University of Chicago, in order to investigate the relative susceptibility of the adrenals to metastasis in comparison with other organs having a similar blood supply. He states that he has found no previous discussion of this subject in the literature. In this series of cases he found records of 821 malignant neoplasms, in 445 of which metastatic deposits had occurred in other organs. In 34% of cases these deposits were present in the liver, in 27% in the lungs, in 13% in the adrenals, in 6% in the kidneys, and in 3·7% in the spleen. He remarks that it is noteworthy that despite the smaller size of the adrenals, more metastatic deposits occur in these glands than in the kidney or the spleen. The incidence of metastases in the adrenals is 25% of all organically metastasizing tumours, and is higher per unit weight than that of liver, lungs, spleen, thyreoid or kidney. As many of the metastatic deposits occurring in the adrenals originate from lung tumours, from breast tumours or from malignant melanomata, it is reasonable to surmise that implantation is by means of the blood stream. They are more frequently bilateral than unilateral, but when unilateral occur more often in the left than the right adrenal gland.

Interalveolar Communications in Normal and Pathological Mammalian Lungs.

The question as to whether communications between alveoli (alveolar pores) normally exist in mammalian lungs has been the subject of a great deal of investigation and discussion since as far back as 1822. C. G. Loosli (*Archives of Pathology*, December, 1937) has devised a new method of investigation by filling the lungs with a stainable substance so that the relationship of the pores to the other constituents of the alveolar walls could be studied. This was accomplished by injecting intratracheally into the lungs of rabbits and rats heparinized blood plasma. This was allowed to clot, after which the lungs were removed and sections were prepared. In the sections the alveoli were seen to be filled with a fibrin network and strands of fibrin could be traced through the alveolar pores in the walls of adjacent alveoli. The pores in each alveolus varied in number from one to eight, and in diameter were from five to thirteen microns. They are well displayed in photomicrographs and *camera-lucida* drawings accompanying the article. The alveolar pores were also studied and demon-

strated in the lungs of dogs and monkeys rendered pneumonic and killed at different stages of the disease. The author concludes from these observations that the interalveolar openings are part of the normal structure of the alveolar walls and that they are one of the important avenues of intralobar spread of infection in experimental lobar pneumonia. The fact that the pores are of normal occurrence seems to be further evidence that the lining of the alveoli is composed of an amorphous membrane of mesenchymal origin over the surface of which are scattered single nucleated cells which may be endodermal or mesodermal in origin.

MORPHOLOGY.

The Interosseous Muscles of the Hand.

C. R. SALSBURY (*Journal of Anatomy*, April, 1937) gives an account of a study of the interossei of thirty hands. This study brought out certain points not in agreement with the usual textbook description. The palmar interossei are inserted, with few exceptions, wholly into the extensor expansions. The first dorsal interosseous muscle is inserted wholly into the proximal phalanx. The other three of the dorsal group have a variable insertion, more frequently into both extensor expansion and proximal phalanx. As a rule these two insertions are from functionally separate fleshy portions, and this separation may be so complete as to form an extra palmar interosseous muscle. The second dorsal interosseous muscle has a greater insertion into the proximal phalanx than has the fourth, and the fourth has more than the third. Several minor anatomical features are described and an attempt is made to correlate structure and function.

Structure of the Junction between the Nerve Root and the Central Nervous System.

I. M. TARLOV (*Archives of Neurology and Psychiatry*, Volume XXXVII, 1937, page 555) gives a description of the nature of the junction between the central and peripheral nervous system. The central glial segment of a nerve presents essentially the structure of a fibre tract of the brain, its peripheral segment corresponding to a peripheral nerve. The homology between oligodendrocytes and Schwann cells, suggested by Cajal and ably supported by del Rio-Hortega, is emphasized by the similarity in structure and by the occurrence of oligodendrocytes in the peripheral nerve segment replacing Schwann cells for a short distance. Transitions between the two types of cells occur. That oligodendrocytes may have a function other than their relation to myelinization is indicated by their occurrence along the course of the central segment of the olfactory nerve, in which region myelin cannot usually be stained. Ganglion cells occur frequently along the peripheral course of motor nerves and are more commonly seen on the sensory nerves in the outlying regions of their ganglia. Islands of glial tissue occur rather commonly in the peripheral nerve segment. They have been particularly striking in the case of the eighth nerve, which contains a longer glial segment than any other cerebrospinal nerve.

The Sheath of the Rectus Abdominis Muscle.

ROBERT WALMSLEY (*Journal of Anatomy*, April, 1937) states that developmentally the sheath of the *rectus abdominis* muscle is a true rectus sheath derived from the rectus mass, but as the lateral muscles obtain attachment to it, it assumes an aponeurotic character. The sheath gains its final significance only when it is considered in mechanical terms of the muscle resultants of the lateral muscles. The typical portion of the rectus sheath exists in three main forms, each form being an expression of the action of the lateral abdominal muscles. The atypical form of the lower part of the rectus sheath is due to a difference in the function of the lower parts of the abdominal muscles. The arcuate line (*linea semicircularis*) is the lower edge of the upper actively contracting parts of the abdominal muscles. The sheath of the rectus, even in its variations, remains the expression of the action of the lateral muscles.

The Nerve Supply of Bone.

DANIEL J. HURRELL (*Journal of Anatomy*, October, 1937) gives a description of the nerves observed in preparation of bone. Nerve fibres were traced into and along the Haversian canals of adult bone and into the bone matrix. Their distribution, characters and endings in the bone matrix are described. Some fibres end blindly in the bone matrix, some in close relation with the bone cells. The author suggests tentatively that the nerve fibres found may be the two ends of a reflex arc covering bone growth and maintenance.

Abnormal Bifurcation of Aorta.

G. I. BOYD (*Journal of Anatomy*, April, 1937) describes a somewhat rare condition discovered in the dissecting room. The aorta bifurcated at the level of the third lumbar vertebra, the right common iliac artery passing to the right under cover of the inferior *vena cava*, and the left common iliac artery being a continuation of the aorta. The right common iliac artery was constricted by a fibrous slip from the right crus of the diaphragm, which passed down in front of it to the disk between the third, fourth and fifth lumbar vertebræ. It was then crossed by the right lumbar sympathetic trunk and passed downwards and backwards into the psoas muscle, which formed a tendinous arch over it. The abnormal common iliac artery emerged from the psoas through another tendinous arch opposite the disk, between the fourth and fifth lumbar vertebræ, and ran down on the right side of the commencement of the inferior *vena cava*. Two figures are given.

Cyclic Morphological Variations in the Anterior Hypophysis.

C. S. CHADWICK (*American Journal of Anatomy*, November, 1936) made a study both quantitatively and qualitatively of the hypophyses of eighty-eight normal, sexually mature guinea-pigs. He demonstrates that cyclic variations occur in the percentage of the cell types in the anterior hypophysis of the guinea pig and, further, that these variations may be correlated with the periods of the œstrous cycle. In general these changes are: a high level of chromophile cell types during the follicular phase of the cycle and a low level of chromophile cells during the lutein phase. With respect to each chromophile type, the basophile cells are high in relative percentage in pro-œstrus and undergo degranulation early in the œstrous period. The eosinophile cells reach their peak in early œstrus and undergo degranulation in late œstrus and met-œstrus. Both types remain at a low level during the lutein phase and begin to climb when follicular influence is again manifest over the accessory organs. A basic intra-alveolar colloid has been described as characteristic of the anterior lobe of the hypophysis, and evidence has been presented to show that it increases in amount with increasing body and pituitary weights.

Innervation of the Intrinsic Muscles of the Eye.

SAM L. CLARKE (*Journal of Comparative Neurology*, Volume LXVI, 1936, page 307) reports experiments which in his opinion show that the intrinsic muscles of the cat's eye are well supplied with motor nerve fibres, each muscle fibre apparently being related to at least one nerve termination. The ciliary muscle and the sphincter of the pupil are supplied from the ciliary ganglion; the dilator muscle of the pupil receives its fibres from the superior cervical sympathetic ganglion. No positive evidence of proprioceptive innervation of these muscles was seen. No evidence has been found that the terminal fibres of the nerves supplying this smooth muscle arise from an anastomosing nerve net. Though many endings outside of the muscle cells were observed, no intraprotoplasmic nerve terminations in smooth muscle cells were seen; but this should not be taken as proof that intraprotoplasmic endings do not exist.

British Medical Association News.

SCIENTIFIC.

A MEETING of the Queensland Branch of the British Medical Association was held at the Brisbane Hospital, Brisbane, on November 5, 1937. The meeting took the form of a series of demonstrations by members of the honorary staff.

Recurrent Hæmatemesis.

DR. D. A. A. DAVIS showed a male patient, aged forty-four years. By occupation he was a packer in a grocery firm; there was nothing noteworthy in his family history and he had had no serious illness or operation.

On admission to hospital he complained of attacks of epigastric pain for the past three or four weeks, the pain not being related to meals and being sometimes followed by vomiting. No loss of weight or indigestion had occurred until four weeks previously; there was no history of jaundice or of either constipation or diarrhœa, and there were no symptoms of any urinary disability. There was no cough.

Physical examination revealed a thin, pale-looking man, moving restlessly in bed. Some pyorrhœa was present, and the left tonsil was enlarged. Examination of the heart and lungs revealed no abnormality. On examination of the abdomen he complained of tenderness under the right costal margin; no definite rigidity or masses were felt. The reflexes and reactions of the pupil were normal. A Wassermann test of his blood, made since his discharge to the out-patient department, gave no reaction.

The day after his admission to hospital his abdominal discomfort became worse and he was given morphine and atropine, and was placed on Hirst's diet, first period. A blood count revealed 4,500,000 red cells per cubic millimetre, with a colour index of 0·9, and 11,000 leucocytes per cubic millimetre. The next day he had an attack of acute epigastric pain, and a subsequent Graham's test revealed a pathological condition of the gall-bladder. Prior to this two carious teeth were extracted, and four days later, after the extraction of two more teeth, he had a large hæmatemesis and became pulseless and very restless. The usual treatment by morphine, ice by mouth and rectal injections of saline solution was given. On the following day he vomited some coffee-ground material, but no bright blood. Two days later he had a further large hæmatemesis. Ten cubic centimetres of calcium gluconate were given, followed by a blood transfusion from a suitable donor, twenty ounces of citrated blood being given, also eight cubic centimetres of 1% Congo red intravenously. However, at 1 a.m. the next day his colour became very poor, his lips were cyanosed, and a further blood transfusion of sixteen ounces of blood over a period of forty minutes was given that day, followed by twenty ounces of blood given over a period of fifty minutes. At 9.30 a.m. the next day he had improved, but was not so well again later in the afternoon. A blood count then revealed 2,030,000 red cells per cubic millimetre and a hæmoglobin value of 35%. Two days later there was still melena present, and a further fainting attack occurred. The systolic blood pressure was found to be 82 millimetres of mercury and the diastolic blood pressure 30. Twenty cubic centimetres of 10% calcium gluconate solution were given intravenously and twenty cubic centimetres of "Campolon" intramuscularly; also a further transfusion of eighteen ounces of citrated blood was given over a period of fifty minutes. Oxygen was also continuously administered by the nasal catheter. His blood urea was found to be 121 milligrammes of urea per 100 cubic centimetres of blood, on account of dehydration. The blood sugar was also estimated and was found to be 0·138%. From that time, however, the patient began to improve, and five days later was having a liberal carbohydrate diet and thirty grains of iron and ammonium citrate. He continued to improve, and ten days after his last hæmorrhage his blood urea was found to be 33 milligrammes per 100 cubic centimetres of blood, and his blood count revealed 2,420,000 red cells per cubic millimetre, with a hæmoglobin value of 46%; there were 7,000 leucocytes per cubic millimetre; anisocytosis, poikilocytosis and polychromasia were present. He was now allowed certain vegetables and eggs, and his diet was gradually increased to include fish, chicken or lamb, and the usual articles of a hospital "light diet".

A blood count two weeks later revealed 4,100,000 red cells per cubic millimetre; the hæmoglobin value was 80% and the colour index was 0·9; the leucocytes numbered 7,400 per cubic millimetre. There was slight anisocytosis and the platelets were normal. He complained of constipation, nausea and abdominal pain, and was passing small constipated tarry stools. He also vomited some bile. However, these symptoms and signs gradually cleared up, and ten days later he was discharged to the out-patient department after seven and a half weeks in hospital, during which time he had two large hæmatemeses, two small ones and further hæmorrhages by the bowel, and received four blood transfusions over a period of four days. Each transfusion improved his general condition temporarily, but it was not until some time after his fourth transfusion that he really could be taken off the dangerously ill list. At the time of the meeting he stated that he felt splendid, had put on weight, ate everything and did his usual work without any trouble.

Dr. Davis said that the diagnosis appeared to be an acute gastric ulcer due to possible septic foci in teeth and gall-bladder, and he considered that it would probably be advisable to take a swabbing from the patient's gums, make an autogenous vaccine, and give him a course of this before extracting any more teeth. Also, a further Graham's test, a fractional test meal and a barium meal seemed indicated.

Post-Rubella Encephalitis.

DR. N. W. MARKWELL showed a patient, a married woman aged thirty-seven years, whom he had first seen on September 24, 1937, when she complained of a sore throat and generalized pains, and said she was feeling ill. Her temperature was 38·35° C. (101° F.), the pulse rate was 100 per minute, and the respirations were 20 per minute. Five days before the patient was first seen she had had rubella, but had been better for two or three days. She was very "run down"; she was still suckling her fourth child twice in twenty-four hours, and had had her second bout of lobar pneumonia since the birth of that child. The temperature, pulse and respiration rates gradually came to normal in the next three days, and the patient seemed much better. However, instead of continuing to improve, she became progressively weaker, and within five days of the post-rubella rise of temperature the temperature, pulse rate and respiration rate dramatically dropped within twenty-four hours to 35° C. (95° F.), 54 per minute and 14 per minute respectively. At the same time the patient looked very toxic, and the muscles of the whole body were very feeble, but there was no definite paralysis. On the next day the patient began to vomit and vomited uncontrollably for nearly forty-eight hours. She was unable to pass urine, and it was impossible to move the bowels even with repeated enemata.

This was one of the two first cases in Brisbane of post-rubella encephalitis, and it was just at the period of the acute anterior poliomyelitis scare. Dr. Lilley and Dr. Earnshaw both concurred in the diagnosis of myelitis, but Dr. Gutteridge found that the cell count in the cerebro-spinal fluid was one only. There was no rigidity of the back, and the patient could kiss her knees quite well; the cerebro-spinal fluid was not under increased pressure and there was no blood in it. Dr. Earnshaw gave the opinion that unless the myelitis was so severe that no cell reaction could occur, the case was not one of Heine-Medin's disease. There was marked retraction of the abdomen; when vomiting ceased, a left-sided facial palsy developed, followed the next day by a right-sided facial palsy. The patient meanwhile was feeling better, and she was able to pass urine and her bowels were opened naturally. However, during the night of onset of the right-sided facial

palsy she had definite respiratory distress and was taken to the Brisbane Hospital to be handy to the artificial respirator. She had some respiratory distress for the next two nights, but this was not very severe. She gradually improved and the power began to increase in the muscles of her limbs and body. Two weeks previous to the meeting power had returned in the right side of the face, to be followed by partial return on the left side, but she still could not move the lower lid of her left eye at all, and there was a possibility of residual paralysis there.

Most text-books stated that there were no complications of rubella, but complications comparable with those in ordinary measles had been described in Osler and McCrae's "System of Medicine". Dr. Markwell pointed out that since this case there had been several other cases of post-rubella encephalomyelitis described in Brisbane; these had occurred at the end of a very extensive epidemic. He wondered about the dangers of Parkinsonism following on post-rubella encephalomyelitis; it had been found that after *encephalitis lethargica* the patients who were most likely to develop Parkinsonism were those who had not been kept long in bed. Dr. Markwell proposed to keep this patient in bed for three months.

Purpura Hæmorrhagica.

Dr. Markwell's second patient was a girl, aged twenty-one years, who had been shown to the Brisbane Hospital Clinical Society, since when she had been under observation by Dr. Sippe and Dr. Markwell. On September 15, 1933, she had attended the out-patient department, complaining that she bruised easily and had epistaxis. Her menstrual periods were normal; she had exhibited purpura then and there was a history of recurrences of the condition in childhood. Her blood count had revealed 4,150,000 red cells per cubic millimetre, with a hæmoglobin value of 82% and a colour index of 1·0. The white cells numbered 8,000 per cubic millimetre, 65% being neutrophile cells and 34% being lymphocytes; the platelets numbered 4,150, the normal being 250,000 to 300,000. The bleeding time was six minutes, and the result of the coagulability test four minutes, these being fairly normal. The patient was a typist, working all the time. Four years previously she had given a history of not liking vegetables, and had had no vegetables or fruit for the previous four years. She was told to eat vegetables and was given "Radiostoleum", no vitamin *C* being then obtainable. On November 5, 1933, the platelets numbered 43,000; on February 9, 1934, they totalled 281,820, which was normal; in April, 1934, they numbered 319,000, and in May, 1934, 126,000. In August, 1934, they were normal in number, and after this the patient was not seen till August, 1935, when the platelets numbered 294,000.

Dr. Markwell said that the patient was then lost sight of for two years, when she suffered from epistaxis following German measles and when he saw her. She had also had hæmorrhagic purpura with a number of petechiæ, and the platelets numbered 7,650. She was later admitted to hospital with a platelet count of 4,100. Five cubic centimetres of her own blood were injected into the buttock daily for seven days, and the platelets had risen to only 7,600. On November 1, 1937, the red cells numbered 4,700,000 per cubic millimetre, the hæmoglobin value was 92%, and the colour index was 0·9; the white cells numbered 8,200 per cubic millimetre, neutrophile cells comprising 61%, lymphocytes 35% and eosinophile cells 4%, while the platelets numbered 3,000. If there had been a larger percentage of eosinophile cells, Dr. Markwell said that he might have been encouraged to go on with shock treatment; twenty cubic centimetres of blood was a recommended dose. In the meantime the patient had put on one pound in weight and was back at work. The question of splenectomy arose, and Dr. Sippe advocated this procedure. The patient's sister had been typed and was found to be a satisfactory donor, and a transfusion could be given at any time. This would probably be done before splenectomy was performed.

Dr. Markwell said that Bray, in his recent text-book on allergy, had said that there were two classes of purpura, and that those with a low platelet count were not due to allergy, and therefore injections of whole blood were probably not of much use. The use of ultra-violet rays had been recommended by Osler and McCrae, but had not been tried in this case.

A discussion followed as to the use of snake venom of the coagulative type in these cases, and also the use of Congo red intravenously to increase the platelet count.

DR. C. H. SIPPE stated that lately the patient had developed menorrhagia and more petechiæ; she was probably on the verge of a serious hæmorrhage, and splenectomy must be considered.

DR. NEVILLE SUTTON discussed splenectomy, but stated that all the patients on whom he had operated had been in a very much worse physical condition than this patient. He would be very loath to perform splenectomy on this patient, who appeared to be in perfect health except for the purpura, which was always present when the platelet count was reduced.

Intermittent Claudication.

Dr. Markwell's third patient was a male, aged fifty years. Dr. Markwell said that intermittent claudication was a symptom, not a disease, and might be due to several causes, for example, to arteriosclerosis (*dysbasia angiosclerotica*), to Buerger's disease and to syphilis. Many treatments were advocated, such as stripping of the arteries, the giving of erythrol nitrate, shock therapy *et cetera*; but none of these were of much use. This patient had had cerebral thrombosis in 1936 with hemiplegia and his arteriosclerosis could be regarded as an established fact. He had been suffering such intense pain in the calves of his legs after walking a hundred yards that he had been contemplating suicide. After thirty applications of short wave therapy with the General Electric Company's inductotherm he could walk about half a mile before the onset of pain, which was now bearable. He had then not been seen for six weeks and had relapsed somewhat. After four applications of short wave therapy in a week he could again walk a half mile, whereas after the six weeks without treatment he could walk only two hundred yards without pain. The *dorsalis pedis* artery could be felt beating.

DR. NEVILLE SUTTON discussed the tests for showing dilatation of the peripheral circulation in the disease, and the treatment following on these results.

Coronary Occlusion.

Dr. Markwell's last patient was a male, seventy-six years of age. This patient had suffered from an atypical coronary occlusion and was being shown as a clinical pitfall. He had been admitted to hospital with auricular fibrillation, being so ill that he was put on the dangerously ill list at once. However, the heart rate was only 100 beats per minute and the pulse rate 84 per minute; the auricular fibrillation was obvious, but could not have been causing the disability. The patient had been quite well and then had collapsed suddenly with the onset of the irregular rhythm, feeling very ill. The diagnosis was almost certainly acute coronary occlusion, the auricular fibrillation being but an incident. The auricular fibrillation stopped in two to three days and the waves of the electrocardiograph became typical of coronary occlusion. Another unusual feature was that when the regular rhythm returned, the *P* waves were upside-down. Dr. Markwell thought the infarct must have in part attacked the auricle. If in an aged person the heart symptoms were out of all proportion to the heart signs, coronary occlusion must always be considered, especially if there had been a sudden change. The patient's pulse was now 85 to 90 beats per minute, and was regular.

Conservative Mastoid Operation.

DR. WALTER CROSSE showed five patients in whom atticoantrotomy had been performed. All patients showed cure of suppuration, the ears being dry; the hearing was as before in two cases, and was improved in three cases.

The first patient was a woman aged twenty-seven years, who had first consulted Dr. Crosse on January 15, 1931, complaining that she had not heard with the left ear for as long as she could remember. She had had discharge and deafness in the right ear for three months following a sore throat. Her general health was good. On examination the left ear was found to be stone deaf. In the right ear there was fairly good hearing, a whisper being heard at nine feet, and fork 32 being heard. The result of the Rinné test was positive. There was a perforation in Shrapnel's membrane, with some granulations and a discharge of thin pus; the rest of the drum appeared healthy. The naso-pharynx was healthy; the tonsils were large and unhealthy. Nothing abnormal was detected in the nose and sinuses. The patient's tonsils were removed and regular cleansing treatment to the granulations with *Spiritus Vini Rectificatus* and silver nitrate was carried out from time to time until April 14, 1936, when the patient complained of some ache in the right ear and in the side of the head. She was extremely worried, as her hearing in this ear had gone off a good deal; she could hear a loud voice at two feet. There was some increase of discharge. In view of the fact that her left ear was stone deaf, Dr. Crosse performed an attico-antrotomy operation on her right ear on April 15, 1936. The operation revealed some chronic mastoiditis and cholesteatomatous débris in the attic. By July 16, 1936, the wound was healed and the ear was dry, and the patient could hear a conversational voice at a distance of twelve feet.

Dr. Crosse's second patient was a male aged twenty-three years, who had first consulted him on January 22, 1926, complaining of continuous discharge in the left ear since infancy, the discharge always being worse when he had a cold. The right ear also discharged, but only when he had a cold. He had had two operations for the removal of his tonsils and adenoids, and his general health was good. Examination showed that his hearing was quite good in both ears, but he could hear fork 64 only in both ears, and the watch at a distance of six inches from the left and one foot from the right ear. There was a dry perforation in Shrapnel's membrane in the right ear, the rest of the drum appearing healthy. The left ear contained foul-smelling pus in the meatal canal, with a perforation in Shrapnel's membrane, the rest of the drum appearing normal.

In January, 1926, Dr. Crosse performed a conservative Heath's mastoid operation on the left ear, which revealed chronic mastoiditis. There was some diminution in the amount of discharge after this operation, but the ear still required daily mopping and occasional syringing out. On April 17, 1936, Dr. Crosse performed an attico-antrotomy operation on the left ear. There was some cholesteatomatous débris in the attic. This operation was performed through the meatus. By May 2, 1936, the wound was healed and dry, and the attic and aditus were well epithelialized. The patient said his head felt clearer and he was hearing much better since the operation. The ear had remained dry ever since. Unfortunately the patient had had continuous discharge from his right ear, and in view of the excellent result obtained with the left ear the patient was very anxious to have the same operation performed on the right ear.

Dr. Crosse's next patient was a woman, aged twenty-one years, who had first consulted him on April 21, 1937, complaining of foul-smelling continuous discharge from the right ear for seven years, with some dulness of hearing. She had tried all kinds of drops and syringing, but the ear still continued to discharge. Both tonsils had been removed in 1935 and her general health was good. On examination the left ear was found to be normal. In the right ear, when the hearing tests were made there was a Weber reaction to the right and no response was obtained to the Rinné test. A conversational voice was heard at a distance of one foot, and in the lower tones fork 64 was heard. The upper tones were normal. There was foul-smelling pus in the meatal canal, with a perforation in Shrapnel's membrane. The rest of the drum was healthy looking. On May 24, 1937, Dr. Crosse performed an attico-antrotomy operation which revealed chronic mastoiditis and granulations and débris in the region of the attic. On July 8, 1937, the wound was healed and dry and the attic and the aditus were well epithelialized. The hearing was exactly as before.

Dr. Crosse's fourth patient was a woman aged thirty-six years, who had first consulted him on August 26, 1937, complaining of foul-smelling continuous discharge from the left ear for four years. She had tried all kinds of drops, syringing *et cetera* with no effect. The right ear was normal and the general health was good. Examination revealed foul-smelling pus in the left meatal canal with a perforation in Shrapnel's membrane; the rest of the drum appeared somewhat sclerosed. Hearing tests revealed a Webber reaction to the left and no reaction to the Rinné test. The lower tones were heard for fork 128, and some slight reduction in the upper tones was observed. The conversational voice was heard at a distance of six inches.

On September 13, 1937, Dr. Crosse performed an attico-antrotomy operation, which revealed chronic mastoiditis and cholesteatomatous débris in the attic. On October 19, 1937, the wound was healed and dry and the attic and aditus were well epithelialized. The patient could hear fork 32 and a moderate voice within nine inches; she felt very well and clear in the head.

The fifth patient shown by Dr. Crosse was a woman aged twenty-three years, who had first consulted Dr. Crosse on July 27, 1937, complaining of foul-smelling continuous discharge from the left ear for three years. She also complained of pains at the back of the head, in the left side of the head, and in the temporal region. She stated that she could hear well with the left ear. She had tried all kinds of drops and syringing, but the discharge had continued from the left ear. Her tonsils and adenoids had been removed eighteen months previously and her general health was good. On examination foul-smelling pus was found in the meatal canal, with a perforation in Shrapnel's membrane; the rest of the drum appeared healthy. Hearing tests revealed a Weber reaction to the left; fork 32 was heard and the response to the Rinné test was positive. The upper tone limit was normal and the conversational voice was heard at two feet. On October 14, 1937, an attico-antrotomy operation was performed; a sclerotic type of mastoid was found, with cholesteatomatous débris in the antrum and attic. By November 5, 1937, the wound was healing well, the attic and aditus were epithelialized and there was no discharge; the mastoid cavity was healing well. The hearing had improved and the conversational voice could be heard at ten feet. The patient said she had no headaches and felt well.

Foreign Body in the Lung.

Dr. Crosse also showed a woman aged twenty-five years, who had consulted him first on October 22, 1937. She gave a history of having had some teeth extracted under a general anæsthetic three weeks prior to that date. When she recovered from the anæsthetic she complained of pain in the right side of her chest, and three days later she became very ill, her temperature rising to 39·45° C. (103° F.). An irritable cough was present, and she brought up a lot of foul-smelling mucopus. On October 23, 1937, a bronchoscopic examination was made, which revealed in the right bronchus much mucopus that was removed by suction aspiration. A portion of a stopped molar tooth was located in the right inferior lobe bronchus, corking this branch. An X ray picture taken before the bronchoscopic examination showed a portion of tooth to be lodged in the right main bronchus. It had evidently tracked down to the inferior lobe bronchus after the picture had been taken. On the tooth being dislodged with the bronchoscopic forceps a bad odour was immediately noticed. As the portion of tooth was too large to pass through the bronchoscope, the bronchoscope, forceps and tooth were all brought up together. The bronchoscope was then reinserted and the bronchi of the right lung were thoroughly aspirated. Dr. Crosse said that the patient had progressed very satisfactorily.

Fractured Neck of the Femur Treated with Well-Leg Traction Splint.

Dr. Harold Crawford showed a patient to demonstrate a treatment which could be used in cases of fracture of the femur in the region of the hip-joint. This type of splint was of benefit in aged patients prone to respiratory complications. The patient in that splint could be sat up in bed and out of bed. Many of these patients were not suitable for treatment by the introduction of the Smith-Petersen nail.

Dr. Crawford said that apart from nailing or the use of such apparatus there were three forms of treatment commonly used. The first was skin traction, which did not control external rotation. The second method was plaster, but elderly patients encased in a Whitman's plaster were very prone to respiratory complications and pressure sores. The third method was to leave the patient without support and to get him up as soon as possible. This was a negative form of treatment, and caused a great deal of pain and resulting deformity. The splint Dr. Crawford was showing was made locally to his own design, but the "push-and-pull" principle was first made use of by Jones and Anderson in the splints constructed by them. Through a movable and a rigid arm traction was made on the affected limb by contra-push on the sound side. This caused tilting of the pelvis and consequent abduction at the hip. External rotation was controlled by a lock-nut attached to the footpiece on the affected side. A Kirschner's pin was put through the lower end of the tibia and fibula on the affected side, and this was attached to the splint and bound on with plaster extending above the knee. The sole of the foot on this side was well padded with felt and rubber. By the use of a thumb-screw gradual extension could be made.

Dr. Crawford showed a male patient, aged seventy-four years, who on September 2, 1937, had fallen onto his left hip and was unable to walk because of the pain. On admission to hospital he was found to have pain in the left hip joint on all movements of the lower limb at this joint. An X ray examination revealed a fracture of the neck of the left femur with displacement upwards of the lower fragment and external rotation and abduction of the shaft. The patient was suffering a great deal of pain. Skin traction was applied, but he developed severe bronchitis. The pain continued, the deformity could not be corrected, and a pressure sore rapidly developed on the back. Dr. Crawford saw the patient on September 15, 1937. On September 17, local anæsthesia being used, he inserted a Kirschner's wire through the lower end of the left tibia and fibula and applied the splint. The patient was immediately placed in the sitting position in bed, and five days later was lifted out onto a chair. The reduction of the fracture was good, and callus formation was proceeding satisfactorily. The patient had suffered no discomfort which could not be relieved by posture, and the pressure sore had practically healed.

Hip Flexion Deformity and Tendon Transplantation.

Dr. Crawford next discussed hip flexion deformity. He said that recently Fitchett and Legg had demonstrated the importance of contraction of the *fascia lata* in the production of deformities about the hip joint, especially when patients suffering from paralysis were unsupported. It was characteristic of this deformity that extension at the hip could be obtained with the limb in the abducted position and not when it was adducted. For relief, complete division of the ilio-tibial band below should be made and the *fascia lata* should be separated from the lateral aspect of the femur. Extension at the hip with the limb in the abducted position could be immediately carried out if this was the cause of the deformity.

Dr. Crawford showed a patient to demonstrate two of the most satisfactory types of tendon transplantation that could be performed in cases of paralysis: (i) that of the *tensor fasciæ latæ* through its fascial tendon into the patella, and (ii) that of the *peroneus longus* into the internal cuneiform. The tendon was separated well up and brought through the anterior compartment of the leg.

The patient, a girl aged sixteen years, gave a history that at the age of two years she had suffered from infantile paralysis, after which she was left with paralysis of the left leg. She had had an operation for stabilization of the left foot performed ten years previously. On her admission to the hospital on March 17, 1937, paralysis of the left lower limb was noted. The hip had a flexion deformity of 45°, which, however, could be corrected when the limb was abducted. The power of abduction was poor, and there was no power of adduction. The extensors of the knee were completely paralysed, while the flexors were acting fairly strongly. The knee showed a flexion deformity of 50°. The foot was in an extreme position of equinus, with the flexors of the toes acting feebly, while the power of the peronei was medium. The patient walked in a doubled-up position, with her hand supporting the left knee. On March 19, 1938, an operation for lengthening of the *tendo Achillis* was performed and the *fascia lata* was divided and transplanted into the patella. Immediately after this operation passive extension of the hip in the abducted position was possible. On April 2 a plaster was applied to the knee, and with wedges this was gradually brought straight. The process was slow, and at one stage there were symptoms of stretching of the peroneal nerve. On August 6 the tendon of the *peroneus longus* was transplanted through the anterior compartment into the internal cuneiform. The present condition of this patient was satisfactory; she had good and full movements at the hip joint, except voluntary adduction, which was absent. The knee could be brought straight; the *tensor fasciæ latæ* was acting well as an extensor at this joint. At the ankle joint the equinus deformity had been corrected; the *peroneus longus* had taken the place of the *tibialis anterior* and was gradually increasing in strength. Owing to the pull of the structures on the lateral aspect of the limb during the growth period, there was some *genu valgum* present. After correction of this deformity by osteotomy Dr. Crawford anticipated the fitting of a walking caliper, which had been impossible with the deformities previously existing. The lumbar lordosis associated with the hip flexion had also been corrected.

Actinomycosis.

Dr. Neville G. Sutton showed under the microscope specimens from a case of actinomycosis.

NOMINATIONS AND ELECTIONS.

The undermentioned have applied for election as members of the New South Wales Branch of the British Medical Association:

Abbott, Terence Kingsmill, M.B., B.S., 1938 (Univ. Sydney), Sydney Hospital, Sydney.

Banks, John Mathew, M.B., B.S., 1938 (Univ. Sydney), Sydney Hospital, Sydney.

The undermentioned has applied for election as a member of the Western Australian Branch of the British Medical Association:

Ferris, Richard Dyason, M.B., B.S., 1936 (Univ. Melbourne), Armadale.

The undermentioned have been elected members of the Victorian Branch of the British Medical Association:

Cole, John Bazil, L.R.C.P. *et* S. (Edinburgh), L.R.F.P.S. (Glasgow), 1928, Reid Street, Wangaratta.

Horan, Joseph Anthony, M.B., B.S., 1936 (Univ. Sydney), Saint Vincent's Hospital, Fitzroy, N.6.

Pyman, Clive Francis Henry, M.B., B.S., 1937 (Univ. Melbourne), 8, Lockhart Street, Caulfield, S.E.8.

Rosenthal, Martin Werner, M.D. (Hamburg), L.R.C.P. *et* S. (Edinburgh), L.R.F.P. *et* S. (Glasgow), 1937, 86, Park Street, South Yarra, S.E.1.

Wedlick, Phyllis Thornton, M.B., B.S., 1931 (Univ. Melbourne), Prince Henry's Hospital, St. Kilda Road, Melbourne, S.C.1.

Day, Arthur Frederick Cephas, M.B. *et* Ch.B., 1918 (Univ. Melbourne), Whittlesea.

King, William Ernest, M.B., B.S., 1937 (Univ. Melbourne), Royal Melbourne Hospital, Melbourne, C.1.

Tonkin, Raymond George, M.B., B.S., 1937 (Univ. Adelaide), Base Hospital, Hamilton.

Medical Societies.

MELBOURNE PÆDIATRIC SOCIETY.

A MEETING of the Melbourne Pædiatric Society was held at the Children's Hospital, Carlton, Melbourne, on October 13, 1937, DR. ROBERT SOUTHBY, the President, in the chair. The meeting took the form of a number of clinical demonstrations by members of the society.

Fracture of Malgaigne.

DR. J. B. COLQUHOUN showed a boy, aged ten years, who had fallen from a horse on May 13, 1937, and injured his arm, which had become very swollen about the elbow joint. In a skiagram taken shortly afterwards the doctor had recognized a fracture of the ulna at the junction of the proximal and middle thirds with radial deviation of the proximal fragments, but had not seen any abnormality of the joint. Several attempts at reduction of the ulnar deformity had proved unsuccessful, and on May 23 the arm was put up in plaster. On June 18 the head of the radius or some *myositis ossificans* was felt over the radial side of the elbow joint, and another skiagram of the joint was regarded as normal in appearance. On the patient's admission to hospital on June 22, the left arm was in a plaster cast with the elbow flexed at a right angle, and there was very definite limitation of movement at the elbow joint. On June 25 Dr. Colquhoun had carried out an open reduction of the fracture. The proximal fragment was found embedded in the flexor muscles and there was an attempted union by soft callus of the distal fragment to the posterior aspect of the proximal ulnar fragment. The fracture was reduced with difficulty and showed no tendency to displacement. The head of the radius appeared to resume its normal position and full range of movement could be obtained at the elbow joint. The arm was immobilized in a plaster slab with the elbow flexed and the forearm supinated. Later the slab was removed and active movement was encouraged.

Dr. Colquhoun said that he had shown the patient for a number of reasons. When a fracture of the proximal third of the ulnar with displacement was seen, dislocation of the head of the radius should be suspected. The fracture should be accurately reduced as soon as possible; if attempts by closed methods were unsuccessful, an open reduction should be resorted to without delay. If reduction was obtained the head of the radius would be found to be reduced also, and if the elbow was immobilized in flexion it would usually remain reduced. Dr. Colquhoun wondered whether anyone would suggest that he should make a further attempt to reduce the head of the radius, which was still displaced, and he would like to know if anyone had cut down on the head of the radius in a similar case to discover the reason for dislocation. He said that he had formed the impression that the attachment of the orbicular ligament was torn off from the ulna, and not, as was commonly believed, that the orbicular ligament was ruptured or that the radial head slipped out of this ligament. He would like to expose the head and neck of the radius soon after the injury on another occasion when he got the opportunity, but he would welcome information from anyone who had carried out this procedure. The boy could extend the elbow only to within ten degrees of full extension and could flex it to within ten degrees of full flexion.

DR. H. C. COLVILLE said that he was very interested in the patient shown by Dr. Colquhoun. It illustrated one of the greatest pitfalls encountered in the traumatic surgery of children: the dislocation was liable to be overlooked not only by the surgeon, but by the radiologist. If they remembered that it was quite uncommon to find ulnar injuries alone in children, this mistake would seldom be made. The line of force had to be transmitted through the radius, and if it produced fracture of the ulna something had to happen to the radius first.

In this injury the radial head was displaced by being dislocated anteriorly into the antecubital fossa and then displaced upwards in association with the overlapping of the ulnar fragments. If, owing to the interposition of muscle tissue, the ulnar fracture could not be reduced, Dr. Colville advocated that the surgeon should cut down onto the ulnar fracture and get end-to-end apposition; the radial head, when pushed back, readily slipped into position, but would pop out again with ease. If the arm was put up in full flexion at once, the radial head must keep in place. Dr. Colville had found the treatment he had outlined satisfactory if it could be applied soon after the injury had happened. In the child shown by Dr. Colquhoun, Dr. Colville thought that the problem of the radial head was almost insuperable. Had the arm been kept up for a long time in full flexion the radial head might have stayed reduced. He would advise that even at this late stage a further attempt to replace the radial head should be made. He had found that he had been able to reduce a similar dislocation by manipulation several weeks after the injury. If it could be reduced by traction and backward pressure and if the elbow was then put in full flexion, it might remain in the normal position.

DR. W. FORSTER said that he thought it was particularly difficult to decide whether the annular ligament of the radius was lacerated, torn or displaced.

DR. H. D. STEPHENS said that the ligament was sometimes torn in half and the ends were frayed so badly that it was very hard to get sutures to hold in it. He had seen it suggested that a fascial strip could be fixed to secure the head of the bone. In the patient shown by Dr. Colquhoun he would be inclined to try again to replace the radial head at an open operation.

Dr. Colquhoun thanked for their advice those who had contributed to the discussion. He had tried manipulation repeatedly without success. The radial head had popped into proper position at the open operation and he had put the arm up in very acute flexion, though he had taken care that the radial pulse was not obliterated. He had kept the arm in flexion while in plaster for six weeks, but when movement was begun the radial head popped out. If the ligament was pulled off the ulna, any result he was likely to obtain at another operation was not likely to be better than the present one. On two occasions he had assisted a surgeon who had attempted to use fascial ligaments, but both attempts were unsuccessful. He repeated the necessity to suspect forward dislocation of the head of the radius in these cases, and to secure in the first place good antero-posterior and lateral skiagrams. He considered that success must always be doubtful when the orbicular ligament was ruptured.

Secondary Suture of the Common Peroneal Nerve.

Dr. Colquhoun also showed a male patient, aged ten years, who had been admitted to hospital on March 22, 1937. Eight weeks before his admission the child had cut both legs posteriorly with a cross-cut saw; the wounds on the left leg were deeper than those on the right. The wounds had healed, but when the boy had started to walk after three weeks in bed it was noticed that he had definite left-sided foot-drop.

On his admission to hospital scars were present on the posterior aspect of both legs distal to the knee joints; there was no tenderness, but on the left leg there was

some retraction of the scar. There was foot-drop and absence of voluntary power in the dorsiflexors and evertors of the foot. Sensation over the lateral aspect of the foot and the toes was absent. Operation was carried out on March 31 under general anæsthesia and with the use of a tourniquet. An incision was made along the course of the nerve and the nerve was exposed and found to be damaged about 2·5 centimetres (one inch) proximal to the division into the superficial and deep peroneal branches. There was incomplete division of the nerve, the ends being connected by an attenuated fibrous band; proximal to this band the nerve was bulbous in shape and somewhat thickened, while the distal portion was rounded but not thickened. Guide sutures were applied with fine linen thread and extensive neurolysis was done. Dr. Colquhoun said that both the nerve ends were trimmed by section until they appeared to be normal macroscopically. In this way about 1·875 centimetres (three-quarters of an inch) of nerve tissue were removed. By flexing the knee to its maximum extent it was possible to suture the nerve without tension; the skin wound was closed and the limb was immobilized in plaster of Paris with the knee fully flexed and the foot in the equinus position.

Dr. Colquhoun remarked that the post-operative course had been uneventful; after four weeks the plaster had been removed and the sutures cut, and new plaster had been applied with 50° of flexion at the knee joint. On May 18 the plaster was removed and a long double iron splint with drop stops to prevent stretching of the paralysed muscles had been applied. Ten days later there had been no response to galvanism or faradism in the dorsiflexors or peroneals. On June 22, Tinel's sign was elicited by tapping over the superficial peroneal nerve distal to the neck of the fibula; a tingling had been felt in the foot, especially on the lateral side, and the boy had complained of pain when the nerve was tapped. On September 28 the boy had completely recovered sensation and voluntary power in the muscles supplied by the common peroneal nerve.

Dr. Colquhoun said that he was showing the patient because he illustrated that secondary suture of the common peroneal nerve was worth while. He said that his own experience was somewhat limited, but he had seen several cases which had not been successful. He thought that there were two important points to observe: (i) that suture of this nerve as a primary suture when a clean wound was being dealt with offered a reasonable prospect of success, provided that the muscles supplied by the nerve were not over-stretched in the after-care period, and (ii) that if the wound was infected it was better to leave the nerve unsutured and to perform a secondary suture operation when the inflammation had disappeared. He said that it had been found during and after the War that success could be obtained even two or three years after the nerve had been injured. He thought that in adults return of function might take from one to one and a half years, whereas in children it was much quicker; in the patient he had shown, for instance, it had taken only six months. In suturing nerves great delicacy should be observed, and fine materials were essential. He did not think that chromicized catgut should be employed, but 160 linen thread would be found satisfactory. He considered that meticulous after-care was the secret of success, provided that a reasonable suture operation had been performed.

Dr. H. D. Stephens said that he was satisfied that the patient shown by Dr. Colquhoun would make a satisfactory recovery; it had been a revelation to him that the boy had been able gradually to straighten the knee. Two or three years earlier Dr. Stephens had had a female patient who had had two or three inches of the right external popliteal nerve completely destroyed in a street accident; he had found the nerve to be frayed and had sutured the ends together with the knee fully flexed. In spite of this procedure the patient had not improved, and he had operated again without success; the patient was left with foot-drop, and a stabilization operation was carried out later. Dr. Stephens mentioned that there was a boy in the hospital at the time of the meeting suffering from osteomyelitis of the fibula. At operation Dr. Stephens had found the nerve over the centre of the field of operation, and after the operation the boy had peroneal paralysis. A period of twelve weeks had elapsed and recovery of power was just commencing.

Dr. W. Forster said that five years earlier a child patient of his had been shot in the leg and the sciatic nerve was perforated by the bullet; the boy had complete paralysis in the distribution of the nerve. At operation a week later he had found much scar tissue, in which the severed ends of the nerve were embedded. He had sutured the nerve and wrapped it in a sheath of fascia. Recovery of power was good, with the exception of that of some of the small muscles of the toes. At the time of the meeting the patient was still suffering from intense neuralgia and found it necessary to cut the toes out of the shoes he wore on the foot of the affected leg.

Dr. Colquhoun said that in the short period of six months that had elapsed since the injury the patient he had shown had regained the power to dorsiflex and to evert the foot; three months after the operation the muscles had not responded to galvanism or to faradism. In similar lesions the stage reached might take a year or a year and a half, but it was known that recovery was made more rapidly in childhood than in later life. It was gratifying to note that sensation was almost completely restored.

(To be continued.)

Correspondence.

A DISCLAIMER.

Sir: Referring to the article by Mr. J. G. Whitaker, F.R.C.S., on "The Treatment of Tuberculous Hip" in your journal of March 19, the writer wishes to draw attention to a sentence in which ". . . Mr. C. H. Osborn, now unfortunately no longer with us . . ." appears.

From these words a misconception may arise, and in fact has arisen, to correct which I should like to follow the example of Mark Twain and say that the report of my death has been greatly exaggerated.

Yours, etc.,

Charles H. Osborn, F.R.C.S.

32, Collins Street,
Melbourne,
March 29, 1938.

Obituary.

JOHN LELEAN SCHOLES.

We regret to announce the death of Dr. John Lelean Scholes, which occurred on March 22, 1938, at Ballarat, Victoria.

WILLIAM CUNNINGHAM DENNISTON.

We regret to announce the death of Dr. William Cunningham Denniston, which occurred on March 29, 1938, at Sydney, New South Wales.

Books Received.

A SHORT TEXTBOOK OF MIDWIFERY, by G. F. Gibberd, M.B., M.S., F.R.C.S., M.C.O.G.; 1938. London: J. and A. Churchill Limited. Demy 8vo, pp. 536, with 187 illustrations. Price: 15s. net.

Diary for the Month.

APR. 12.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
APR. 19.—New South Wales Branch, B.M.A.: Ethics Committee.
APR. 20.—Western Australian Branch, B.M.A.: Branch.
APR. 21.—New South Wales Branch, B.M.A.: Clinical Meeting.
APR. 22.—Queensland Branch, B.M.A.: Council.
APR. 27.—Victorian Branch, B.M.A.: Council.
APR. 26.—New South Wales Branch, B.M.A.: Medical Politics Committee.
APR. 28.—South Australian Branch, B.M.A.: Branch.
APR. 28.—New South Wales Branch, B.M.A.: Branch.
MAY 3.—New South Wales Branch, B.M.A.: Organization and Science Committee.
MAY 4.—Victorian Branch, B.M.A.: Branch.
MAY 4.—Western Australian Branch, B.M.A.: Council.
MAY 5.—South Australian Branch, B.M.A.: Council.
MAY 6.—Queensland Branch, B.M.A.: Branch.

Medical Appointments.

Dr. G. T. Gibson has been appointed Honorary Medical Officer at the Wallaroo Hospital, South Australia.

* * *

Dr. C. A. Hogg has been appointed a Member of the Board of Official Visitors to Mount Saint Margaret, Ryde, New South Wales.

* * *

Dr. J. H. Kelly has been appointed, under the provisions of the *Workers' Compensation Acts*, a Certifying Medical Practitioner and Medical Referee at Melbourne, Victoria.

* * *

Dr. T. M. Greenaway has been appointed a Member of the Board of Official Visitors to Bay View House, Cook's River, New South Wales.

* * *

Dr. W. Christie has been appointed, pursuant to the provisions of the *Education Act*, 1915, of South Australia, a Member of the Advisory Council of Education.

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Dr. G. S. Adam has been appointed Superintendent of the Women's Hospital, Brisbane, and Professor of Obstetrics in the University of Queensland.

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Dr. P. F. V. Crowe has been appointed Senior Assistant Medical Superintendent at the Hospital for the Insane, Goodna, in accordance with the provisions of *The Insanity Acts*, 1884 *to* 1935, and the *Public Service Acts*, 1922 *to* 1924, of Queensland.

* * *

Dr. L. A. Windsor-MacLean has been appointed, under the provisions of *The Workers' Compensation (Lead Poisoning, Mount Isa) Act of* 1933, of Queensland, Chairman and Member of the Medical Board constituted under that act.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser", pages xviii-xx.

AUSTIN HOSPITAL FOR CANCER AND CHRONIC DISEASES, HEIDELBERG, VICTORIA: Honorary Officers.
ROYAL HOBART HOSPITAL, TASMANIA: Resident Medical Officer.
THE PRINCE HENRY HOSPITAL, SYDNEY, NEW SOUTH WALES: Honorary Officers.
THE QUEEN'S (MATERNITY) HOME INCORPORATED, ADELAIDE, SOUTH AUSTRALIA: Resident House Surgeon.
VICTORIAN EYE AND EAR HOSPITAL, MELBOURNE, VICTORIA: Resident Surgeons.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment referred to in the following table without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCHES.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company Limited. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. House, 225, Wickham Terrace, Brisbane, B.17.	Brisbane Associate Friendly Societies' Medical Institute. Proserpine District Hospital. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.
SOUTH AUSTRALIAN: Secretary, 178, North Terrace, Adelaide.	All Lodge appointments in South Australia. All contract Practice Appointments in South Australia.
WESTERN AUSTRALIAN: Honorary Secretary, 205, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.

Editorial Notices.

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